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LECTURE II.*

MR. PRESIDENT AND FELLOWS,—In my first lecture I described the clinical features of the condition to which the term "cœliac disease" has been applied, and as a working hypothesis I assumed that the syndrome of symptoms to which Gee gave the name is actually a specific entity. I propose now to consider its pathology, and at the outset I must crave indulgence for the speculative character of much that I have to say.

MORBID ANATOMY.

As evidence of specificity one naturally turns to morbid anatomy and to bacteriology, and, failing these, one seeks a specific cause in disturbance of function in some particular organ or organs. How far is it possible to demonstrate by such inquiry that in so-called "cœliac disease" we are dealing with a morbid entity due always and only to one specific cause?

I must first consider its morbid anatomy, and, unfortunately, the available records and observations are meagre in the extreme.

Dr. Gee himself, in his original paper on the subject, says: "Naked-eye examination of dead bodies throws no light upon the nature of cœliac affection. Nothing unnatural can be seen in the stomach, intestine, or other digestive organs. Whether atrophy of the glandular crypts of the intestines be ever or always present I cannot tell."

Dr. R. A. Gibbons, also, writing a year later (1889) than Dr. Gee, says: "Examination of the bowel after death has led to the discovery of nothing."

The only detailed record of a post-mortem examination which I have been able to find is that published by Drs. Poynton, Armstrong, and Nabarro (1913) in the Proceedings of the Royal Society of Medicine.

It refers to a boy who had had a prolonged attack of diarrhœa lasting eight months in the second year of his life, and again at five years began to suffer with diarrhœa which recurred at intervals until his death at the age of 9 years. The stools were at times of the pale, creamy, offensive character which suggests cœliac disease, the abdomen was much distended, and the boy much wasted. His weight at 8½ years was 27 lb. No record was kept of his height, but he was, as I remember—for he was under my care repeatedly at a convalescent home—much below the normal height for his age. Eventually an attack of vomiting, with severe abdominal pain, for which no cause could be found at autopsy, ended in collapse and death.

The noticeable features at post-mortem were thickening of the walls of the whole intestine both large and small, the mucosa of which shared in the thickening and showed also patches of acute congestion; the solitary follicles were prominent in the large intestine, Peyer's patches in the small were not enlarged, the stomach wall and mucosa showed similar thickening and congestion.

The liver was large, pale, and soft, and showed marked fatty change. There was no obvious naked-eye alteration in the pancreas; there was no tuberculosis anywhere.

Microscopic examination showed much small round-cell infiltration of the mucosa and submucosa, but more in the stomach and small intestine than in the large bowel. In places there were small blood extravasations in the mucosa. The liver showed fatty degeneration. The pancreas showed "marked increase in the interlobular fibrous tissue, particularly that surrounding the ducts," indicating, it was thought, some interlobular pancreatitis.

In connexion with this case I will mention one which showed post-mortem appearances so closely similar that they point almost certainly to the same condition.

Clinically the diagnosis had been in question on account of the unusually rapid course and also the comparatively late age at which the symptoms began. A boy aged 7 years was under my care 19 weeks with looseness of the bowels, amounting at times to actual diarrhœa. He had been sent into hospital as a case of tubercular peritonitis on account of wasting and enlargement of the abdomen, but one early began to doubt this diagnosis, for the stools at times were suggestive of cœliac disease, and, moreover, there was, with œdema and purpura, frequently recurring tetany.

Examination of the stools showed no dysentery bacillus, but a large predominance of a bacillus which could not be exactly identified but resembled in many respects the *Bacillus lactis aerogenes*. On the ground that the case was probably one of cœliac disease the boy, after several weeks of unsuccessful treatment, was put upon a fat-free diet and at once improved greatly, as cœliac cases usually do upon such feeding, but a few weeks later had a relapse of looseness with much distension of the bowel, and died of exhaustion.

At post-mortem there was about half a pint of serum in the abdomen and the bowels presented a remarkable appearance; they were of a dark slaty colour externally, and looked, and were, thickened throughout; the stomach did not show this appearance. On opening the bowel the mucosa was seen to have a swollen and congested appearance throughout, but there was no ulceration.

The liver weighed 19 oz. in spite of being considerably congested; the normal average weight at this age is 23·6 oz., so that the findings in this case confirmed the results of clinical examination by percussion, to which I drew attention in my previous lecture. The pancreas weighed 1 oz. and showed nothing abnormal to the naked eye. There was no tubercle anywhere.

Microscopic examination of the duodenum, ileum, and colon showed much small round-cell infiltration of the mucosa and submucosa, and this was most marked in the duodenum. The liver showed engorgement with blood, but otherwise nothing abnormal. The pancreas showed some excess of fibrous tissue, especially around the ducts, the appearance being suggestive of some pancreatitis.

There was thus, except in the absence in this case of fatty change in the liver, a close agreement in the post-mortem findings in these two cases. Unfortunately scrapings were not taken from the mucosa of the bowel, so that its bacteriology remains doubtful.

It is clear that in these two cases there was an inflammation involving the whole intestinal tract, an inflammation obvious to the naked eye as well as to microscopic examination; moreover, the marked thickening of the wall of the bowel was something quite unusual.

I have examined large numbers of children who have died of diarrhœa, in some cases of many weeks' or months' duration, and in many of very acute intensity, but these ordinary cases of gastro-enteritis or ileocolitis show no such thickening of the bowel; often, indeed, the wall looks rather thinner than normal, as if it were wasted.

I lay stress on this point because it suggests that the inflammatory change was, at any rate, not merely a terminal occurrence; it had probably existed a considerable time, possibly months. On the other hand, it would not be safe to conclude that this was necessarily a primary manifestation, or, indeed, even an essential part of the disease; it is, at least, conceivable that the whole inflammatory process might be secondary to some functional failure of digestive secretions, with resulting abnormal decomposition of food residue favouring the growth of harmful bacteria, and so producing an inflammation entirely secondary in character in the wall of the intestine.

In this connexion I would recall the clinical observation which I mentioned in my previous lecture, that the character of the stools tends to change as the disease progresses.

The pale, porridge-like, rancid stools which are a feature of the earlier stage are often replaced after several months by darker brownish stools with much mucus, perhaps even a little blood, and with an odour offensive enough, but no longer typically rancid. In other words, the stools, which at first were compatible with a fault of digestion alone, at a later stage show clear evidence of inflammatory change in the bowel.

The same difficulty arises in regard to the changes found in the pancreas—Are they primary or secondary?

It is tempting to think of them as primary, as then it would be easy to explain the early occurrence of the pale fatty stools; but again the possibility, if not the probability, must be admitted that the increase of fibrous tissue, the pancreatitis if it be so, may be merely secondary.

BACTERIOLOGY.

If the morbid anatomy, so far as it has been observed, affords no reliable clue to the specific cause of this affection, it gives at least ground for conjecture that some specific bacterial agent may explain the appearances described in the bowel, and though, as I have suggested, these may be secondary rather than primary, the finding of an organism constantly, even where these secondary changes had already occurred, would be presumptive evidence that the organism found was the primary cause of the disease; at any rate, it would give good ground for seeking further evidence of its specificity.

The earliest observations of which I am aware are those by Herter (1908). He studied the fæces of these cases, which he called "intestinal infantilism," and considered that the most noticeable feature was the predominance of Gram-positive organisms. Two, in particular, he considered to be sufficiently constant and predominant to justify the suggestion that they may be causal—namely, a bacillus which he called *Bacillus infantilis* and the *Bacillus bifidus*. Both, however, are by his own admission found in the stools of healthy nurslings, and some observers have found the *B. bifidus* to be common in the normal stool at this age, and have found both of them also in ordinary cases of infantile diarrhœa.

It is therefore clear that neither of these is specific in the sense that its presence is evidence of the disease, nor even in the sense that if it becomes pathogenic at all it will produce this particular symptom-complex. Indeed, the only evidence of pathogenicity mentioned by Dr. Herter in the case of *B. infantilis* is the experiment on a dog which was fed on milk first sterilised and then inoculated with *B. infantilis*. The animal developed a persistent diarrhœa, which ceased only with the discontinuation of the *B. infantilis* milk. A second trial gave a similar result. Injected into guinea pigs, neither *B. bifidus* nor *B. infantilis* showed any pathogenicity.

In addition to these organisms various cocci were found in the stools, but no causal influence is claimed for them.

As confirming the pathogenic rôle of the *B. infantilis* and the *B. bifidus*, Dr. Herter points out that these organisms become less and less noticeable in the stools as the child improves, when such organisms as *B. coli* and *B. lactis aerogenes*, Gram-negative forms, take their place.

*Lecture I. was published in THE LANCET of August 10th, 1918, p. 163.

With these observations are to be contrasted those recorded by Drs. Poynton, Armstrong, and Nabarro in the paper already mentioned.

In their fatal case the stools were examined during life. It is to be noted that the boy was 8½ years old and had been suffering from this affection at least 3½ years when the stools were first examined bacteriologically; they showed predominance of Gram-negative bacilli, some Gram-positive diphtheroid bacilli, and Gram-positive micrococci. Plate cultures showed only Gram-negative bacilli in abundance, and subcultures gave most of the reactions typical of the *B. coli* group. At the autopsy scrapings of the mucosa from the large intestine were used to make cultures on neutral-red agar plates. Coliform and streptococcal colonies were found, but the organism to which special attention was directed proved to be a dysentery bacillus of the Flexner type modified, a Gram-negative bacillus agglutinating with a *B. dysenteriæ Y* agglutinating serum up to 1 in 2000.

In another of their cases bacteriological examination of the stool showed no dysentery bacilli; there were many Gram-negative bacilli and a few Gram-positive streptococci; in this case no opportunity occurred for taking scrapings from the intestinal mucosa.

Bacteriological investigations, for which I am indebted chiefly to Dr. D. N. Nabarro and to Dr. W. d'Este Emery, were made in 11 of my cases.

In three of these the stools only were examined and the dysentery bacillus was isolated from one, a girl aged 2 years, in whom the disease had lasted five months.

In six an agglutination test, as well as a bacteriological examination of the stools, was done, and in four of these the agglutination test gave a positive result with one or more strains of dysentery bacillus (in three almost equally strongly with the Flexner, Shiga, and Y strains); in one of these four a dysentery bacillus of the Flexner type was found in the stools; in two of the six cases where the double investigation was made both agglutination and fæcal examinations were negative as regards the dysentery bacillus. In two cases the agglutination test only was used; one was strongly positive for the Flexner and Y strains of dysentery bacilli and only less strong for the Shiga, the other was negative for all these. These results are shown in the accompanying table:—

Dysentery Bacillus in Cœliac Disease.

Case.	Agglutination test.			Fæces.
	Flexner.	Shiga.	Y	
1	Strong.	Strong.	Strong.	Negative.
2	,,	,,	,,	,,
3	Negative.	Negative.	Negative.	—
4	,,	,,	,,	Negative.
5	Strong.	Partial.	Strong.	—
6	—	—	—	Positive.
7	—	—	—	Negative.
8	Partial.	—	Strong.	Positive.
9	—	—	Partial.	Negative.
10	—	—	—	,,
11	Negative.	Negative.	Negative.	,,

It will be seen, therefore, that out of the 11 cases no less than 6 gave evidence of infection with a dysentery bacillus.

In the two cases in which the stools only were examined and no dysentery organism was isolated the findings were:

In the one, a boy aged 2 years, in whom the disease had been present three months, a predominance of Gram-positive organisms, especially *B. bifidus*, together with streptococci, *B. coli*, and pneumo-bacilli.

In the other, a girl who died at the age of 4 years, after suffering with this disease for nearly three years, the stool a few weeks before death showed Gram-negative organisms predominant, mostly *B. coli*, no dysentery bacillus nor other non-lactose fermenters, a few streptococci, *B. bifidus* in small numbers, whereas about six weeks earlier the Gram-positive organisms, chiefly bacilli, had predominated, and the *B. aerogenes capsulatus* had been present in large numbers; on that occasion, as later, the dysentery bacillus was not found.

The bacteriological findings, it must be admitted, are far from conclusive of any specific infective cause. The most suggestive part is the finding in some cases of a dysentery bacillus, and perhaps the more significance is to be attached to this inasmuch as the serum of some cases was found to agglutinate this bacillus.

The apportionment of pathogenic influence must always be difficult in the face of such a plethora of bacteria as is shown by investigations of the intestinal flora in any particular case or group of cases, but the observations are probably still less conclusive when they are limited to examination of the fæces. More reliable conclusions can be drawn from investigation of the mucosa itself or of scrapings taken from it. An example of this is the discrepancy mentioned in the case quoted above where examination of the stools during life showed no dysentery bacillus, whereas this was isolated in cultures from scrapings of the mucosa taken after death. Herter also found that the *B. bifidus* was much more numerous in the mucus immediately in contact with the mucosa than it was in the fæces. While, therefore, negative results in the bacteriology of the fæces have a certain value they must be received with caution, especially where, as in several of my own cases, only one or two examinations were made. For this reason the agglutination test may be regarded as more reliable.

I think, however, that one must be careful not to attach too much importance even to the agglutination test until much more numerous observations are available, and in particular until similar observations have been made upon cases in the early stages of the disease.

All the cases in which I have mentioned the finding of the dysentery bacillus in the stools, or a positive result for the dysentery organism with the agglutination test, have been cases of at least several months' duration, the earliest fully three months, and some as long as three years.

Before we can regard the dysentery organisms or any other as the specific cause of this affection it is necessary to exclude the possibility that their presence may be a mere epi-phenomenon.

Agglutination, no doubt, goes some way towards this; it proves that the presence of the particular organism concerned is exerting some pathogenic influence on its host, but this does not prove that it is the primary pathogenic influence. The growth and the predominance of this or that organism depends largely upon the soil, and probably there is no part of the body where the soil varies so much as in the intestine, but its variations in health are, so to speak, within fixed limits and dependent partly upon the character of the ingesta and partly upon digestive processes.

It is known that with temporary disturbances of digestion there are alterations, if not in kind, certainly in the relative proportions of the intestinal bacteria, and it is therefore conceivable that with profound and prolonged disturbance of digestive processes the soil may be so altered that an organism like the dysentery bacillus, which is now known to be much more widely disseminated than was thought a few years ago, might find a particularly favourable nidus and add its pathogenic effects to those of the primary digestive trouble.

I suggest this only as a possibility. There is clearly a strong probability that in some of the cases of cœliac disease, if not in all, there is a primary infective catarrh of the bowel, and that this is due, in some at least, to the dysentery bacillus.

FAILURE OF FAT ASSIMILATION.

I turn now to another question which calls for consideration in seeking for the primary and essential cause of this disease. Is it possible that the *fons et origo* is some profound digestive disturbance and not infective at all? As I pointed out in describing the clinical symptoms, one of the most characteristic phenomena of cœliac disease is the pale stool, with appearance and smell suggestive of excess of fat.

As a homely method of demonstrating this, I have observed the sooty, spluttering flame when a match dipped in these fæces is set alight. Microscopically, also, it is evident in some of the stools by the large excess of fatty acid crystals, and sometimes by the obvious excess of fat globules, but the only certain gauge is quantitative chemical analysis.

According to Dr. Cammidge,[9] the normal range of variation on an ordinary mixed diet is 10–25 per cent. of fat in the stool, whereas in cœliac disease Herter found in one case (a boy, aged 8 years, in whom the disease had lasted five years) the total fat in the fæces amounted to 25–47·2 per cent. In one of Dr. Cheadle's cases the fat in the stools varied from 19·8 to 49·65 per cent., and in another was 33 per cent.

Dr. Cammidge has estimated the fat in three of my cases.

1. A girl, aged 5½ years; duration of disease, 3½ years; percentage of fat, 26·9.
2. Boy, aged 18 months; duration of disease, five months; percentage of fat 73·1.
3. Girl, aged 3 years; duration of disease, 12 months; percentage of fat, 42·5.

I have no figures from my own cases to show what proportion the fat passed in the fæces bears to the fat taken in the food—a very important practical point. Herter, however, investigating one of his cases, found that there was a loss of 20–25 per cent., and on one occasion even as much as 40 per cent. of the ingested fat. The normal loss in health varies from 2 to 8 per cent.; the average is 5 per cent.

Failure to absorb the fat of the food may be due to various causes, but traced to their sources these fall into two categories: (1) lack of preparation of the fat for absorption through deficiency of the fat-splitting function of the bile or of the pancreatic secretion; (2) failure of the intestinal surface to absorb, in spite of the fat being sufficiently prepared by the fat-splitting processes and saponification.

Question of Hepatic Insufficiency.

The pale colour of the stools led Dr. Cheadle to suppose that the deficiency was mainly one of bile.

Speaking (1903) of the stools in these cases, which he grouped under the heading acholia, he said: "They are obviously as destitute of bile as those of complete obstructive jaundice." More in accordance with recent observations is Dr. Gee's statement: "The paleness is commonly supposed to signify lack of bile, but the colour of fæces is a very rough measure of the quantity of bile poured into the duodenum, nay, more, stools are not always so wanting in bile as they seem to be, in particular opaque white food such as milk curd," (he might have added, "milk fat") "undigested, will hide the colour of much bile."

Certainly of any organic change in the liver there is no hint in this disorder.

Even in the most severe cases and in the most extreme stage there is no tendency to jaundice, nor has any change been observed post-mortem, except that in one of the cases quoted above the liver was fatty, but such a change is found, presumably as a terminal occurrence, in various conditions where it is certainly no essential part of the disease, for instance, with abdominal tuberculosis.

Analysis of the fæces, however, in two out of three of my own cases showed some deficiency of bile.

Indeed, in the one mentioned above, containing 73·1 per cent. of fat, there was only a trace of stercobilin and no biliverdin. In the other, containing 26·9 per cent. of fat, Dr. Cammidge notes "the reaction for stercobilin is not as well marked as in a normal stool." In both these cases it seems natural to suppose that deficiency of bile is responsible, in part at least, for the failure of fat absorption.

On the other hand, I must mention that in the third case, although there was 42·5 per cent. of fat in the stool, there was no apparent diminution of stercobilin (incidentally, it is noteworthy that the colour of the stool in this case was nearly as pale as porridge, showing that the colour alone is little gauge of the activity of the liver). The deficiency of stercobilin in these cases has been noted also by Dr. Hutchison,[10] but he points out that there is no other evidence of hepatic insufficiency, in particular the urea secretion attains its normal level.

In connexion with this question of hepatic insufficiency I would recall the observations mentioned in my previous lecture showing that the liver dulness in cœliac disease is usually smaller than normal. I know not whether there is any connexion between the smallness of size and the deficiency of secretion, but it is, at least, a curious coincidence.

On the whole, there seems to be evidence that one of the fat-splitting agents, the biliary secretion, is deficient, at least in some cases. I shall consider later how far this is responsible for the failure of fat absorption.

Question of Pancreatic Insufficiency.

The chief agent in the preparation of fat for digestion being the pancreas some fault in this gland or its function has naturally been suspected.

Dr. Gibbons (1889) suggested that cœliac disease might be due to some profound inhibition of the functions of the pancreas. The combination of symptoms also, chronic looseness of bowels with arrest of growth, recalls the cases which Dr. Byrom Bramwell[11] described as "pancreatic infantilism."

But is there any satisfactory evidence of pancreatic insufficiency in these cases of cœliac disease?

Approaching the question first from the clinical side I tried in three cases Sahli's test, administering iodoform in glutoid capsules to see if the capsule, insoluble by the gastric juice, had been dissolved by the pancreatic secretion, so that the drug could be detected in the urine within a few hours after administration. In one only of the three this reaction—i.e., the failure of the drug to appear in the urine—indicated deficiency of pancreatic secretion.

I also tried Loewi's test, dropping adrenalin solution into one eye and taking dilatation of the pupil on that side as an indication of pancreatic insufficiency. In four cases out of six there was no dilatation of the pupil, in two there was slight but quite definite dilatation, and one of these was the case in which Sahli's test was also positive, in the other the Sahli's test was not used.

The microscopic investigation for azotorrhœa, as it has been called—in other words, failure of digestion of muscle fibres, which has been regarded as evidence of imperfect pancreatic digestion—gave negative results in five cases examined.

In five cases the amylase content of the urine was investigated on the lines directed by Dr. Percy Stocks.[12]

In the absence of any sufficient data for children of this age the normality or abnormality of the results could only be judged by applying the same method to control cases of approximately the same age. This was done, and it was found that on the whole there was remarkably little variation amongst the controls, 10 units was the number found in three cases, 6·6 in one, and 20 units in two cases, whilst of cœliac cases one showed 13·3 units, another 10, another 20, and another 6·7, so that in none was there an increase of amylase such as is supposed to indicate affection of the pancreas.

Whether the amylase content of the urine is a reliable index of the sufficiency of the pancreas has yet to be proved. It is difficult to believe that the amount of amylase in the urine can be a safe indication of the efficiency of the pancreas unless the diet is taken into account, for Pavlov has shown how profoundly the ferments of the pancreatic juice can be influenced experimentally by diet.

To sum up the result of my clinical investigations of the pancreatic function in these few cases, there were indications in some that it was defective, but in the majority all the tests applied proved negative.

So far, therefore, as these tests have any weight, the balance of evidence is against any pancreatic insufficiency as a constant or essential factor in the disorder.

There is another clinical observation which may point in the same direction—namely, the failure, at any rate in my hands, of pancreatic extract in treatment, a point of contrast between these cases and the "pancreatic infantilism" in which Dr. Bramwell obtained such rapid and remarkable improvement with preparations of pancreas.

Laboratory investigation of the stools gives but little support to the idea of pancreatic insufficiency.

Indeed, in the three cases in which Dr. Cammidge examined the stools for me, the trypsin index in each was rather unusually high, suggesting that there was even abnormal activity in that gland. In two of these Dr. Cammidge thought that the excess of trypsin was so considerable as to point to some active inflammatory change in the pancreas, an interesting suggestion in view of the post-mortem finding in the cases I have quoted.

Further evidence of pancreatic activity is the character of the fat passed in the stools. Herter's observations showed that the greater parts of the fats lost are in the form of fatty acid, and that the sum of the fatty acids and the soaps make up, on the average, about three-quarters of the fat lost. These results, compared with the normal averages found by Dr. Cammidge (1 per cent. of neutral fat to 20 per cent. of fatty acids and soaps), show a proportion of neutral or unsplit fat which would not be inconsistent with some slight degree of pancreatic insufficiency.

Even this, however, is negatived by the finding in my three cases already mentioned, where the proportion of split fat in the stool was much higher, varying from 25·9 per cent. to 72·1 per cent., as against only 1 per cent. of neutral fat. The percentages will be best seen from the accompanying table, in which the normal is given for comparison:—

—	Total fat.	Neutral fat.	Fatty acids.	Soaps.
Case 1	28·9	1·0	14·6	11·3
Case 2	73·1	1·0	33·8	38·3
Case 3	42·5	1·0	30·1	11·4
Normal, on mixed diet (Cammidge)	21·0	1·0	9·0	11·0

It is evident from these figures that there is no failure in the splitting of fats, and if one assumes that the pancreatic secretion plays the chief part in fat-splitting the idea of any "profound inhibition of the function of the pancreas" is clearly untenable.

Failure of Fat-absorbing Power of Intestinal Mucosa.

It would seem, then, that, although in individual cases the biliary secretion may be found deficient, there is no interference with the fat-splitting process which prepares fat for absorption, and therefore we must conclude that the fault lies in failure of the absorptive function of the intestinal mucosa. Is this a functional failure or is it due to organic change in the intestinal mucosa?

If we could assume as the primary factor in cœliac disease a functional failure of the fat-absorbing power of the intestines, this would account for many of the features of the disease, for, as I have already pointed out, the excess of unabsorbed fat in the stools favours putrefactive changes which in themselves probably render the stool irritating to the bowel and so would account for the looseness of the bowels; moreover, with putrefactive changes there would be alteration in the character of the intestinal flora, and probably conditions favourable to the growth of pathogenic organisms, so that, as a secondary phenomenon, inflammatory lesions would occur.

But apart from the difficulty of proving that purely functional failure of intestinal absorption ever occurs, there is such definite clinical evidence in a certain proportion of cases of cœliac disease that some catarrhal or inflammatory condition is an early, if not the earliest, pathological change that is seems a more probable view that even in the cases which appear to begin quite insidiously there is some degree of catarrh, for these, like the others which start with definite diarrhœa, show before long more or less of the typical recurrent looseness of the bowels.

Gee evidently suspected that there was some organic change in the intestinal mucosa, for he hints, as I have mentioned, "atrophy of the glandular crypts in the intestine," but even in the fatal cases which I have described, in which the disease had lasted in the one five months and in the other at least four years, microscopical examination showed no such atrophy. On the other hand, definite acute inflammatory infiltration was found, but it is extremely doubtful whether the appearances were consistent with inflammation which had lasted years, as it must have done if it is to account for the non-absorption of fat, which is one of the earliest symptoms of cœliac disease.

Moreover, it is difficult to reconcile the presence of an acute inflammation as an essential factor in the disease with the observations of Dr. Gee that the bowel showed nothing abnormal in his case. The clinical facts also are opposed to any acute inflammatory condition as an essential factor, for sometimes the onset is quite insidious and without diarrhœa; on the other hand, the symptoms in the later stages of the cases which do badly, the passing of much mucus and sometimes blood, point unmistakably to some acute irritation, probably inflammatory process, in the bowel.

The first step in the disease may well be a catarrhal condition of the mucosa in the small intestine, where in health the absorption of fat occurs chiefly or entirely.

I suppose it is conceivable that such a catarrh, due to bacterial irritation, might eventually, especially with the constant irritation due to putrefactive change in the unabsorbed fat, become more acute or be altered in character by the supervention or predominance of some particular organism—e.g., the dysentery bacillus.

But though it is agreed that catarrh of the small intestine may interfere with the absorption of fat, it is difficult to see why, if this explanation be the correct one, the failure of fat-absorption should be so much more pronounced a feature in cœliac disease than in other diarrhœal conditions where catarrh of the upper part of the whole of the bowel occurs—for instance, the common summer diarrhœa or other forms of gastro-enteritis.

WASSERMANN REACTION.

In the search for a specific cause of cœliac disease the possibility of congenital syphilis had hardly presented itself to my mind, so conspicuously absent had been all the usual symptoms of such infection.

In the light, however, of recent experience showing how slight or equivocal may be the clinical evidence where a Wassermann test is found to be positive—and one must add that in some such cases, but by no means in all, the diagnosis of syphilis is confirmed by the results of treatment—it seemed advisable to apply this test to cases of cœliac disease. This was done in six cases; in three out of the six the reaction was positive.

It would be out of place here to enter into any general consideration of the significance to be attached to a positive Wassermann reaction obtained where all other evidence of congenital syphilis is lacking, but at the risk of being held guilty of heresy I will venture to say that in my opinion a positive Wassermann reaction, extremely valuable as it is as confirmatory evidence, is not by itself proof of congenital syphilis.

These cases of cœliac disease in which the test was positive were none of them in any way suggestive of congenital syphilis so far as family history or the child's own past history was concerned, nor on examination was there any symptom of congenital syphilis, unless the cœliac disease itself was to be regarded as such. Treatment with mercury in one case, so far from doing any good, seemed rather to hinder improvement; at any rate, the child began to improve directly it was stopped; in another it seemed to have no effect at all.

There seemed, in fact, so little to warrant accepting the reaction as proof of congenital syphilis that we sought corroboration in testing the parents and other members of the family.

In one severe case of cœliac disease, a girl aged 3 years and 5 months, the Wassermann reaction was done twice and was on each occasion strongly positive; the mother and the father were both definitely negative.

Another case, a girl aged 5½ years with moderately severe cœliac disease and showing, as did the previous case, very marked arrest of growth, gave a definite positive Wassermann reaction; her mother gave a negative; the father was abroad and could not be tested.

The third case, a girl aged 5 years and 4 months, a milder case of cœliac disease but showing arrested growth, gave a positive reaction; the father was not available for testing, but the mother and also the sister, 15 months younger than the patient, were both entirely negative.

In none of these cases had the relatives who were tested had any treatment for syphilis so far as was known.

I leave those who consider a positive Wassermann reaction to be *per se* proof of congenital syphilis to explain such cases with what ingenuity they may.

These findings seem to me rather to emphasise the uncertainty of significance attaching to this reaction where there is no other reason to suspect syphilis, and to support the view that there are other conditions beside syphilis which may give a positive result with this test.

TOXIC ABSORPTION.

I shall turn now from the unanswered, and at present unanswerable, questions of pathogeny which I have raised to a consideration from the pathological standpoint of some of the clinical phenomena of cœliac disease. One of the noticeable symptoms, especially in severe cases, is dropsy occurring chiefly in the distal parts of the limbs and also the face, and sometimes associated with ascites.

This is common enough in any prolonged diarrhœa in infancy and, indeed, with any condition of extreme wasting. As it occurs most commonly in advanced stages of disease it is apt to be regarded as chiefly, if not altogether, an evidence of failing circulation, with, perhaps, some hydræmia as a contributory cause.

Without denying that these may play some part, as also may the diminished urinary secretion in diarrhœa with the resulting retention of chlorides, I would point out that there may be another factor, and that the œdema which occurs in cœliac disease may well be determined by it—namely, toxic absorption from the intestine.

Apart from the fact that no particular substance having this effect has been isolated from the intestine or its contents, it may be objected that in cœliac disease wasting and feebleness are usually pronounced, and that therefore there is no reason for seeking further for a cause; at any rate, such a view would require support from cases in which these recognised causes are absent.

There are at least two groups of cases which seem to me to corroborate this suggestion.

First, a very common one in which there is puffiness of the eyelids, especially of the lower eyelid, accompanying chronic constipation or chronic indigestion; this surely is œdema at its vanishing point, or rather its first appearing point. I have thought that among the children brought to one for minor ailments one could often pick out those with chronic intestinal disorder, especially affecting the lower bowel, by this puffiness of the lower eyelids.

The second group of cases, in which clinical association and sequence seem to point to a similar connexion, is a rare condition of general œdema with no albumin in the urine and no evidence of organic disease. Puzzling cases are these, and various are the explanations which have been offered. I am not referring to those in which such a condition follows scarlet fever and is associated with diminution in the secretion of urine without albuminuria, but to children in whom there has been no recent illness, and the general œdema apparently stands by itself, an unexplained phenomenon. In some of these the stools are found to be extremely fœtid and offensive although there is no diarrhœa, rather indeed the reverse; therewith the urine may or may not be diminished in quantity.

An instance of this was a boy, aged 11, who a week before coming under observation was noticed to be puffy in the face, and three days later his legs were swollen; the bowels were open three or four times a day, and the stools were extremely offensive. There was some fluid in the abdomen and general dropsy. The urine contained no albumin, it was high coloured, and rather scanty. The boy was kept under observation, and the outstanding feature, apart from the œdema, was the foul fœtor of the stools.

I can offer no proof that the œdema in such a case is due to toxic absorption from the bowel, but the association suggests it.

How far the œdema which accompanies such a condition as ileo-colitis and prolonged failure of digestion is due to such absorption and how far to the wasting and exhaustion is, I think, open to question, but there is much to suggest that in some of these also the œdema is determined largely by the unwholesome character of the stools. I have particularly noticed that in some wasted infants the appearance and disappearance of œdema corresponded closely with variations in the degree of unwholesomeness of the stools, and seemed to be much more influenced by this than by the number of the stools.

This observation certainly applies to cases of cœliac disease, in which, therefore, I would suggest that œdema is to be regarded as due, in part perhaps to feeble circulation and hydræmia, but in part at least to toxic absorption from the bowel.

Certainly the œdema is not necessarily terminal. I have known it to occur in several cases quite early in the disease, and in those that recovered as well as in those that died.

Another symptom which is worthy of consideration in this connexion is purpura.

This, like œdema, is common enough as a late symptom of many diseases which end with emaciation and exhaustion, but it is particularly noticeable that in cœliac disease purpura is sometimes a recurring symptom for years.

For instance, in a girl who died at the age of 9½ years after suffering with cœliac disease from the age of 20 months, bruise-like patches of purpura appeared on many occasions from the age of 2½ years up to the time of her death, although during the greater part of this time she was well enough to be up and about.

In another case, a boy, aged 7½, in whom the disease had been present for five years, but who was still well enough to get up and walk about, patches of purpura occurred from time to time, and noticeably in relation to unhealthiness of the stool; when the stools improved and became more normal the purpura would cease to appear, then a particularly unhealthy stool, without diarrhœa, would be associated with a fresh patch or two of purpura.

I would lay some stress upon the fact that often, so far from there being any increase of frequency of stools, there has often been a noticeable decrease in their frequency, perhaps no stools at all for 24 hours, just before the purpura appears. The non-frequency of stools may be more than a coincidence; delay in the evacuations of the unhealthy fæces may be a determining cause of the absorption which results in purpura.

But, even if this view be correct, the particular substance which causes the purpura is still to seek.

Mere constipation, even when most chronic and severe, does not cause purpura, although there may be various symptoms suggestive of toxic absorption, headache, lassitude, sallow skin, and so forth; it is probable, therefore, that the cause is to be sought either in the products of particular bacteria or in the chemical products of intestinal putrefaction, and only when these hypothetical substances are present does absorption from the intestine produce purpura.

It has been suggested that indol may be the poison in question; and when indol is being formed in large quantities in the bowel this is indicated by a marked indican reaction in the urine. It has been specially noticed that in many cases of purpura a marked indicanuria is present; in one of my cases of cœliac disease I found a very definite indican reaction during the eruption of purpura, and this reaction became much less, in fact, almost entirely absent, when the purpura ceased to appear.

Apart from œdema and purpura, the occurrence of tetany in cœliac disease probably points to toxic absorption.

Here, at any rate, the question of cause is not complicated by any likelihood of production by feebleness or failing circulation; the tetany may, as in one of my cases of cœliac disease, recur for years, and that when the patient is well enough to be up and about.

It is true that tetany has been attributed to deficiency of calcium salts in the blood; and in cœliac disease there is an obvious reason for excessive loss of calcium. Quite apart from the question of tetany, this drainage of calcium was emphasised by Dr. Herter in connexion with the amount of split fat in the form of soap passed in the fæces. In one case he found that in ten days a boy aged 8 years lost 1·07 g. of calcium oxide in 198 g. of soap in the fæces, and such a loss, though it may sound very small compared with the intake of calcium in the food, is really large in relation to the very small proportion of the food-calcium which can be absorbed in health.

But does this satisfactorily explain the tetany?

I think not. If it were so, one would certainly expect that the occurrence of tetany should correspond more or less closely with the periods when the fatty character of the stools is most in evidence. I have no analytical evidence that the early pale, rancid, obviously fatty stools contain more fat than the later brown slimy or shreddy stools in which excess of fat, if present at all, is certainly not obvious; but if one may assume this, as I think one fairly may, then there is no such correspondence between the tetany and the fatty stools; indeed, the reverse is the case, for tetany is not an early symptom in cœliac disease, it occurs after the disease has been present for a long time and usually when the stools, though extremely offensive, have lost the characteristic fatty appearance of the early stage.

Absorption of some toxic material from the bowel seems much more in harmony with the clinical fact.

ARREST OF GROWTH.

From these questions of toxic absorption I pass to the consideration of one of the most striking phenomena of cœliac disease, the arrest of growth.

Why do these children fail to grow? It is easy to reply vaguely that the cause is the general failure of nutrition, but such an answer is inadequate. It is unfortunate that much that has been written on the problems of growth entirely ignores the fact that increase of weight and length do not necessarily go *pari passu*, and that experimental evidence as to the one is not necessarily applicable to the other. A physiological illustration of this must be recognised by any one who measures many children at the growing age; a rapid increase in height is not associated usually with increase in weight, often, indeed,

with a marked decrease, and the child may be visibly thinner; this is very noticeable in infants and gives rise to much needless anxiety, though I have not seen it mentioned in a single text-book that an infant in perfect health will often cease to gain or will actually lose weight when it is growing rapidly in length.

In disease, also, there is no necessary correlation between the growth in length and the state of general nutrition as shown by the weight. As I have mentioned already a marasmic infant may grow steadily in length, and a child may become tall whilst wasted with chronic tubercular disease.

It would seem, therefore, on grounds of analogy that there is no justification for assuming that the remarkable arrest of growth in length in cœliac disease is a necessary corollary of the arrest of growth in weight, and, while it seems only reasonable to assume that both may be related in some degree—though not necessarily in the same degree—to the general failure of nutrition, the failure of growth in length is so much more pronounced in cœliac disease than in most other conditions of disturbed nutrition that we may well seek for some special cause.

Amongst the various possibilities which present themselves there is one which, I think, ought to be considered—namely, that deficiency of fat absorption may be sufficient cause for arrest of growth.

This suggestion has the support of experimental evidence; young rats fed on a diet of lean horseflesh, so that there was hardly any fat given, became stunted in growth, even though apparently in good health; with a diet exclusively of oxflesh, which, though still affording them too little fat, contains more than does horseflesh, the rats throve better, but their growth was still retarded to some extent. (Watson and Hunter.[13])

If the failure of fat assimilation be the cause of the failure of growth this would bring cœliac disease into line with Byrom Bramwell's pancreatic infantilism, where arrest of growth was associated with the passage of fatty stools and where improvement of fat absorption by administration of pancreatic extract, with consequent disappearance of the fatty character of the stools, was followed by rapid growth.

It is not apparent, however, how deficiency of fat in the diet has any special influence in arresting growth.

If the absorption of calcium is dependent to any considerable extent upon the splitting of fat and combination with the fatty acids thus set free, and if deficient absorption of calcium inhibits growth then the order of events is easy to understand. As I have already pointed out, Herter has already shown that there is an appreciable loss of calcium with the soaped fat passed in the stools of cœliac disease. Clearly the same result would come also from any considerable failure of fat splitting or saponification.

I have mentioned a case in which remarkable bending of the bones began at the age of 8 years, after the cœliac disease had been present nearly five years, and the child had been kept for four years on a diet containing a minimum of fat; an occurrence strongly suggestive of calcium deficiency.

I have shown, also, that there is very striking delay of ossification of epiphyses, which may indicate a deficiency of calcium, but, granted this deficiency, is it adequate cause for the striking arrest of growth in cœliac disease? I have shown in my previous lecture that growth in height shows no necessary relation to the date of commencement, nor to the stage of advancement of ossification in epiphyses.

That relative deficiency of calcium and rapid growth are by no means inconsistent is shown by what happens in cretins when thyroid treatment is begun at the age when walking has recently been acquired, the child begins to grow rapidly and at the same time if the child is allowed to run about much, the previously straight legs become bowed and bandy, presumably from deferred calcification of the rapidly growing bone.

The disease which might have been expected to throw most light upon this point is rickets.

There is strong evidence that fat deficiency in the diet is a large factor, perhaps the determining factor, in the causation of rickets; moreover, there is clear evidence of deficient calcium deposit in the rachitic bone. Here, then, we seem at first sight to have the required link bringing the fat absorption into direct relation with formation of bone.

But if this view of the pathogeny of rickets is correct we are faced by another difficulty in regard to cœliac disease—it should be associated closely with rickets; whereas one of the very striking facts which I have already mentioned in connexion with its symptomatology is the entire absence of rickets in most cases. Conversely also, if the failure of fat absorption be the cause of the stunting in cœliac disease, arrest of growth might be expected far more commonly than it occurs in rickets.

The problem is evidently a complicated one, and it is not rendered less so by a comparison with such a condition as the congenital steatorrhœa described by Garrod and Hurley,[14] in which the passage of 80 per cent. of fat in the fæces, amounting to a loss of 25 per cent. of the fat intake, was associated neither with arrest of growth nor with rickets.

THE LATE JOHN ST. CLAIR BOYD, M.D. R.U.I.—Dr. John St. Clair Boyd, the well-known Belfast surgeon, who died suddenly of cardiac disease, was a graduate of Queen's College, Belfast. After further study at Edinburgh and Paris he became an assistant to Mr. Lawson Tait in Birmingham when that well-known surgeon was at the height of his career. Returning to Belfast, he was appointed surgeon to the Samaritan Hospital, but ill-health obliged him to retire about ten years ago. He was also consulting gynæcologist to the Ulster Hospital for Women. Dr. Boyd was a man of literary taste and well versed in the study of the Irish language. He married a daughter of the late Dr. Stevenson Macadam, lecturer on chemistry in the Edinburgh Extra-Mural School.

THE TREATMENT OF CASES OF SHELL SHOCK IN AN ADVANCED NEUROLOGICAL CENTRE.

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WHILE in charge of an advanced neurological centre in France during the period November, 1916—February, 1918, I have had to deal with between two and three thousand cases of psychoneurosis (neurasthenia, hysteria, and psychasthenia), and in the following paragraphs I will endeavour to give, in broad outline, a general view of the methods of treatment which have seemed to be most useful, and a statement of certain statistical results. The great majority of cases came under my care within 48 hours of their breakdown, and I was able to return 70 per cent. of them to the line after an average of a fortnight's rest and treatment in hospital; of course, certain individual cases needed a longer rest in hospital, up to two or three months. I feel sure that this success was due more often to *prevention* than to cure. By seeing so many of these light cases so early I was able to *prevent* the development of further hysterical or pithiatic symptoms which would otherwise have taken place.

Essential Factors in Causation and Treatment.

It is important that the patient should have the benefit of a thorough examination of his nervous system at the earliest possible moment after the shock of the shell explosion. The incipient functional symptoms from which he is suffering make him fear, vaguely or definitely, that he has sustained some organic injury of the nervous system. This fear encourages the further development of such symptoms. It and its effects can be brought to an end by the reassuring remarks of the neurologist, after he has completed his examination. But those remarks must be repeated and forced upon the patient's notice. Especially is it the note of certainty in the doctor's voice which carries conviction. By one means or another the patient must be completely convinced of the truth of the doctor's explanation of his symptoms, and of the promise that they will quickly disappear. This conviction must pass beyond the stage of mere intellectual awareness and acceptance, and acquire the dynamism of strong emotion. Enthusiastic expectation of a rapid recovery is another essential condition of success, just as it was an earlier apprehension which was responsible for the development of the symptoms.

This is more than mere suggestion and counter-suggestion. The mechanical processes of auto- and hetero-suggestion do certainly come into play in the *fixation* of the symptoms and in their later development and multiplication. But the *origin* of the symptoms (tremors, difficulties of speech and locomotion, profuse sweating, headaches, diminution or loss of various forms of sensation, &c.) is to be found in the intense emotion of fear caused by the shell explosion, of which they are the objective physical manifestations. In like manner, although counter-suggestion plays its part in the cure of these functional symptoms, the factors which are of very much greater efficacy and importance in bringing about a *permanent* cure are: (1) persuasion, whereby the patient is rationally convinced of the true nature of his symptoms; and (2) the sthenic emotions of confidence, conviction, and expectation, which have a unifying effect upon the mind, and counteract the disintegrating effect of mechanical suggestion.

It will thus be seen that I adhere to Déjerine's theory of the pathogeny of the psychoneuroses in preference to that of Babinski. As regards the "period of meditation" which often elapses between the occurence of the emotional shock and the onset of the earliest symptoms, I would also follow Déjerine in holding that it is a period of subconscious emotional development. Babinski and his followers believe that it is a period during which auto- and hetero-suggestion coöperate to bring about the ultimate outbreak of the symptoms. The original shock, no doubt, produces some weakening of mental synthesis, although outwardly the patient does not appear greatly disturbed. The appropriate

emotional reaction, with its innervations and external manifestations, develops gradually during the following few hours or days, as the patient's intellectual awareness of the accident arouses by association earlier emotional memories. The symptoms that eventually arise are such emotional manifestations in a state of relative dissociation and permanence. As Déjerine says:—

"Everything that an emotion may create in an accidental and transient way hysteria may accomplish in a lasting way."[1]

Case of Delayed Onset of Symptoms.

I have several examples of this delayed onset of symptoms in the case of hysterical mutism and hysterical hemiplegia which I hope to analyse and describe on another occasion. But I have obtained the following unbiased introspective description from one of my officer patients who suffered from hysterical spasmodic contractions of the abdominal and leg muscles and profuse sweating and tachycardia, which came on some days after the time when he was exposed to heavy shelling. His account is as follows:—

"My feelings during the shelling are hard to define, as I was too fully occupied to allow for much thought on the subject. Owing to the small area to which we were confined, there was no opportunity of being able to give vent to the pent-up feelings that were in me, and in consequence my nerves were strung up to such a pitch that I felt that something in me would snap. Every shell fired seemed to be nearer the mark than the last, and the ground all around was covered with shell holes. The general feeling was that 'the next one' would land right in the post. Part of the trench had already been blown in. The back blast from each explosion flattened us up against the wall of the trench.

The days following I was always thinking of this episode, and nights I could never sleep, but would just doze and then wake up with a start, with my heart palpitating furiously and with great difficulty in breathing. I would also find myself in a profuse cold sweat, especially of the scalp, forehead, and hands. Then my feet began to be affected and would shake as though I had the ague. This would come on in the day at times, but invariably happened at night when I was lying down. About 10 days later I was troubled with my stomach [he means abdomen]; at night something—I suppose my nerves—started pulling me up forward as I lay down and my body shook all over. This would happen every two or three minutes. This action affected my back, and I had a belt of pain around me. During this time I was always troubled with my heart, and I found it hard to lie down at all. The cold sweats also continued."

Emotional Origin of the Symptoms.

This description, written by an officer unversed in psychology and quite ignorant of the question at issue, is in favour of the emotional origin of the symptoms, although, of course, factors of suggestion are not entirely absent. The emotion of fear in this patient during civil life always produced cold sweats, palpitations, and tremors of the limbs—just as in the case of a patient subsequently suffering from functional paraplegia it used to "take him in the legs" and he would feel weak at the knees. The period of incubation in some cases seems to correspond to the time during which the patient is struggling, with a certain degree of success, to repress the painful emotional memories and to remain "captain of his soul." The final outbreak of the symptoms represents the partial failure of this effort of repression, or rather is a condition of the success of the *psychological* repression. The symptoms are "convertion" symptoms, in Freud's sense of the term—i.e., they represent painful emotion converted into physical innervations.

With a certain class of patients, a more detailed form of mental analysis is necessary. In these cases the nervous breakdown is partly due to earlier mental worry, and the "active service" element is simply the last straw. The patient's mind is a battlefield of conflicting emotional tendencies, many of which he himself fails to recognise. By long talks with him, following up thread after thread in the association of his ideas, one can recall repressed memories of earlier epochs in his life, showing him the true emotional significance of them and their connexion with his present disability. In this way the patient learns to understand himself, and therefore I would call the method "*autognosis*." Self-knowledge brings with it self-control in the psychic domain. An analysis of the patient's dreams is often of the greatest help in this method. Indeed, the dreams of some of these patients are so terrifying that they have to be dealt with, and the quickest way to exorcise them is to analyse them—i.e., to trace the different parts of the dream back to their source in the patient's earlier life (by the method of "free" association), and so to discover the underlying unconscious dream thoughts which have been enjoying a disguised or distorted fulfilment in the dream itself.

[1] Psychoneuroses and Psychotherapy, pp. 269, 270.

Treatment.

I now come to a therapeutic method which I have found to be especially valuable in dealing with *early* cases of shell shock exhibiting pronounced symptoms of functional nervous disorder, such as functional mutism, deafness, paralysis, tremulousness, &c., accompanied by more or less extensive amnesia. It is a form of hypnosis, but free from the defects attaching to the ordinary use of that method. To explain by an example:—

Here is a patient who was blown up and buried by a shell explosion two days ago and lost consciousness. On regaining consciousness some hours later he found that he was quite dumb, and also had lost all recollection of the shell explosion and of the events immediately following thereon. In other words, he is functionally mute and has retrograde amnesia. His memory for other recent experiences is also vague, but he is in full command of gesture-language and can write down on paper all that he wishes to say.

I interview him alone in my office and tell him in a tone of conviction that I shall restore his speech to him within a few minutes if he will do exactly what I say. I then tell him to lie down on a couch, close his eyes, and think of sleep. I urge him *to give himself up to sleep*, to let sleep come to him, as it assuredly will. I tell him that he is getting drowsy, his limbs are getting heavy with sleep, all his muscles are relaxed, he is breathing more and more slowly, more and more deeply. Above all, that his eyelids are getting heavy, as heavy as lead, that he feels disinclined to open them, that he cannot open them however hard he tries. At this stage, which generally supervenes within two or three minutes, he really cannot open his eyes. This is a stage of very light hypnosis quite sufficient for my purpose.

I now tell him that the moment I put my hand upon his forehead he will seem to be back again in the trenches, in the firing line, in the fighting, as the case may be, and will live again through the experiences that he had when the shock occurred. This I say in a tone of absolute conviction, as if there is not the slightest shadow of possibility of my words not coming true. I then place my hand on his forehead. He immediately begins to twist and turn on the couch and shouts out in a terror-stricken voice. He talks as he talked at the time when the shock occurred to him. He really does live again through the experiences of that awful time. Sometimes he speaks as if in dialogue, punctuated with intervals of silence corresponding to the remarks of his interlocutor, like a person speaking at the telephone. At other times he indulges in imprecations and soliloquy. In some cases he is able to reply to my questions and give an account of his experiences. In others he cannot do so, but continues to writhe and talk as if he were still in the throes of the actual experience. *In every case he speaks and acts as if he were again under the influence of the terrifying emotion.* It is as if this emotion had been originally repressed, and the power of speech with it, and is now being worked off and worked out.

Abreaction.

This process of "working off" repressed emotion is what Freud calls "abreaction." In my view it is the essential therapeutic process in dealing with the majority of war psychoneuroses. The original shock caused a *dissociation* of consciousness—i.e., an apparent suppression of a certain series of memories, and of a certain motor function (speech). Now, without accepting Freud's sexual theory of the origin of the psychoneuroses, for which I have never been able to find any convincing evidence (i.e., as a universal pathogenic factor), I *do* accept his theory of the cause of mental dissociation—viz., that it is the result of mental conflict and involves repression of emotional states. The repression of any emotion at its inception involves the danger of dissociation, the ideas accompanying the emotion being then more ready to split off from the rest of the mind and pursue a subconscious life of their own.

In most cases of nervous shock caused by shell explosion a state of intense fear is aroused in the patient's mind, which, from its very magnitude, produces loss of self-control and apparent loss of consciousness. There is no real loss of consciousness, but the attempted repression and control of the fearful emotion at its inception brings about a splitting of the mind, which appears later as an amnesia of greater or less extent, often involving other losses of function also, such as dumbness, deafness, tremulousness, or paralysis. The fact that under light hypnosis, and with the appropriate suggestions, these memories return, together with the lost voice, hearing, &c., is evidence that they were not abolished at the time, but were simply split off from the main personality. It is also evidence that the shock worked mentally rather than by its accompanying *physical* concussion (which is often absent).

In my method, then, the patient goes through his original terrifying experiences again, his memories recurring with hallucinatory vividness. It is this which brings about the return of his powers of speech, and not direct suggestion, as in the ordinary method of hypnosis.

My second modification of the ordinary method is in my manner of awakening the patient. Remembering that his disability is due to a form of dissociation and that in some cases hypnotism accentuates this dissociation, I always suggest at the end of the hypnotic sleep that he will

remember clearly all that has happened to him in this sleep. More than this, I wake him very gradually, talking to him all the time and getting him to answer, passing backwards and forwards from the events of his sleep to the events in the ward, the personalities of the sister, orderly, doctor, and patients—i.e., all the time re-associating or re-synthetising the train of his memories and interests.

By the process of *abreaction* I remove the underlying *cause* of the patient's original dissociation, and in attacking this cause I cannot be accused of merely treating symptoms.

I agree entirely with Pierre Janet that only hysterical patients can be hypnotised—in fact that, as Charcot said many years ago, "hypnosis is an artificial hysteria." It follows from this that as the patient improves under treatment he should become less and less hypnotisable. This is the case with my form of the method. Hypnotism has been brought into disrepute by those seekers after the marvellous who have intentionally split up the personality of their subjects more and more in order to get ever more wonderful and abnormal manifestations from them.

Using this modified form of the hypnotic method, one can abolish that tendency towards "*equivalents*" which Janet mentions as one of the three stigmata of hysteria (the other two being *suggestivity* and *distractivity*). By this is meant the tendency which the patient shows to suffer from another functional symptom as soon as his original symptom is removed by suggestion under hypnosis. For example, a mute may develop headaches or gastric symptoms as soon as he has been made to speak by suggestion under hypnosis. By using "abreaction" this tendency is completely eliminated. The use of suggestion *without* hypnosis quite fails to remove this tendency to develop equivalent symptoms. The equivalent symptoms are often so vague that they are not noticed at the time, although they are none the less disabling.

Results of Treatment.

The hypnotic method is needed only for the very small minority of cases that arrive at an advanced neurological centre—only for those showing so-called major hysterical symptoms. In two series of 1000 cases each I treated 173, or 17·3 per cent., in the first, and 132, or 13·2 per cent., in the second by means of hypnosis. In these two series there were altogether 121 cases of loss of speech, or hysterical mutism. I succeeded in curing *every single one of these cases*. Many of them were deaf as well as dumb. These were given a paper of instructions to read in which they were told to lie down, close their eyes, and give themselves up to sleep. When such a patient showed by the calm look on his face that he had reached a quiet frame of mind, I would suddenly and unexpectedly bang two books together near him, and have the pleasure of seeing his eyes flicker, and then find that he could hear. I could then continue the treatment by word of mouth in the ordinary way. Curiously enough, these deaf-mutes were the easiest of all my cases to treat.

Of course, there are some patients who are ready to malinger with major hysterical symptoms. I caught out 28 such patients in my first series of 1000 cases, and induced them to confess. But this is, happily, a very small proportion—less than 3 per cent.

If, finally, the modified hypnotic method is limited in application, wherever possible, to *one* treatment only, all the drawbacks with which the ordinary hypnotic method is beset are evaded. I only applied the method to the more severe cases, those that had later to be evacuated to the base.

Nevertheless, in such apparently severe cases as those of functional paraplegia this method was not found to be necessary. Vigorous persuasiveness was sufficient to produce a cure. In some of the lighter cases of mutism also this was found sufficient.

Stammering is much more difficult to cure than mutism. The patient can more easily cling to the first symptom than to the second, and every hysteric clings to his symptoms. But in several cases I have produced a complete cure, and a decided improvement in others. In several cases where the patient had stammered in civil life I was able to trace the stammer back to a shock in early life, in the first few years of childhood, and under hypnosis I was able to call up the experiences with hallucinatory vividness before the patient's mind. The stammering was not completely cured thereby, because it had become the habit of a lifetime, but this recall of the shock was the first necessary step towards a cure.

Another Illustrative Case.

As a further illustration of my method I will describe the case of an officer, aged 25 years, whom I treated in France in December, 1916, and whom I happen to have again under my care at Craiglockhart in May, 1918.

He had been sent into my neurological wards on account of his complete inability to stand shell fire, having the irresistible impulse to rush to his dugout and crouch down there the moment shells began to fall in his neighbourhood. Earlier in the year (1916) he had been wounded during an advance and had been left out in No Man's Land under continuous shell fire for several hours. He gave a history of great timorousness and extreme weakness of memory during his youth. I learnt from him that he had been nearly drowned at the age of 3 years, according to what his mother had told him. He himself, of course, could remember nothing of the incident. I should also add that he exhibited many psychasthenic symptoms—e.g., he would begin to count the squares round the top of the stove in his ward, and find that he could not stop counting them. He would be impelled to count them hundreds of times.

I hypnotised this officer and then directed his attention back to the drowning incident at the age of 3 years. He at once began to live again through this incident, gasping with terror as he again, in memory, fell into the water. He described the whole event with such a wealth of detail that it was difficult not to believe that he had been taken straight back to this early period in his life and was living again through the terrifying experience. Later on, he re-experienced with hallucinatory vividness the incident of his baby brother's funeral, seeing again the coffin being lowered into the grave and feeling the desperation of grief *as a present emotion*. He was 18 years old when this funeral took place.

I concluded from these and other results of the hypnosis that the patient's memory was intact, but that certain factors were interfering with the power of recall—viz., the very strong emotional tone of many of these memories, combined with repression of them following upon mental conflict. The "abreaction" of these painful memories in the hypnotic state—i.e., the working off of their emotional accompaniments—should therefore prove beneficial to the patient's powers of memory and to his general mental condition. This was found to be the case, and the patient appeared quite a different, and certainly a much more normal, person after the treatment.

A minor point of interest arising out of this case is that the patient was readily hypnotised in spite of his psychasthenic symptoms. Some authorities have held that psychasthenics are not hypnotisable.

In May of this year I learn from this patient that his mother vouches for the accuracy of his revived memories of the drowning incident in every particular. He is again in hospital suffering from certain neurasthenic symptoms, but he has completely recovered from his previous hysteria. He has done a year's duty since he was with me in France.

Other Treatment.

General hygienic measures are, of course, necessary in the after-care of severe cases, and suffice for the entire treatment of the largely preponderating light cases. Regularity is insisted upon in all habits—alimentation, excretion, sleep, exercise. The patients are put on physical drill and sent for short route marches. Many do light duty in the wards or in the grounds of the hospital. But to the "gospel of work" is added the gospel of cheerfulness and hope.

For insomnia suggestion treatment at night is often very efficacious. The patient can often be soothed to sleep and incidentally taught how to employ auto-suggestion in this case.

Only very rarely have I found it needful to *isolate* patients. But this device succeeded in the case of three patients showing persistent tremors of the head, where every other method had failed.

Summary.

The essential therapeutic agent in the case of *hysteria* caused by shell shock is "abreaction," or the working off of the repressed emotion caused by the shock. Mental analysis is a means to this end, and light hypnosis, applied under proper safeguards, is the quickest and most effective method of effecting this analysis, where amnesia is present and the case is seen early.

For cases of *neurasthenia*, which are the more numerous and involve emotional preoccupation often dating back many years, mental analysis and re-education, without hypnosis in any form, are the needful agents, although the abreaction of the original emotional disturbance or disturbances is again essential.

In both classes of cases the arousing of *sthenic emotion* in the patient's mind is an important adjunct in the cure, both in the form of enthusiastic confidence in his doctor and expectation of a complete recovery, and also in the form of

vivid interest in some form of occupation in the stage of convalescence, during which the mind becomes more unified and consolidated.

I have hitherto not mentioned *rest*, which is, of course, also fundamental. But no complete rest is possible while the mind is obsessed with bottled-up emotion. This emotion must be completely worked off, and then true rest will come. The preoccupations of the neurasthenic also must first be dealt with if any form of rest cure is to produce good results.

After three and a half years of work with nerve patients in military hospitals in Egypt, England, and France, during which over 4000 cases have passed through my hands, I feel no hesitation in saying that mental analysis is the ideal method of treatment, provided that it is carried far enough to produce true abreaction of emotional states. As an aid, and as a sort of short cut, the special form of hypnosis which I have described is very useful in early cases showing major hysterical symptoms.

In conclusion, I should like to thank Lieutenant-Colonel C. S. Myers, F.R.S., R.A.M.C., for his unfailing encouragement and most helpful advice as consultant psychologist in his frequent visits to my wards in France.

May 29th, 1918.

ON THE DIFFERENTIAL DIAGNOSIS OF THE DYSENTERIES:

THE DIAGNOSTIC VALUE OF THE CELL-EXUDATE IN THE STOOLS OF ACUTE AMŒBIC AND BACILLARY DYSENTERY.

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AND
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IN a paper on Dysentery published in 1913 by Savage and one of us,[1] it was pointed out that dysentery, from its first authentic record in the days of the Athenian pestilence during the Peloponnesian War up to the present time, has been *the* scourge of armies in the field, and that it has been responsible for more sickness, invaliding, and military waste than any other factor.

Scope of Investigation.

The greater part of the present paper was written by us in the spring and summer of 1916, and the results to be set forth in the bacillary section were based upon the microscopic and cultural examination of a series of 1165 cases (43 fatal) admitted to a general hospital in Egypt, from April 30th, 1915, to April 30th, 1916. As many stools as possible were plated out on a specially modified MacConkey's medium, and all the fatal cases were submitted to autopsy.

Unfortunately, our collaboration was interrupted by the recall of one of us (C. H. S.) to Government service in Australia, in the late summer of 1916, before we were able, owing to lack of amœbic material, to complete our paper together for publication.*

Since then a further very large series—though we have not the exact figures to hand—has come under our observation. This series included many cases of acute amœbic dysentery, and has been examined by one of us, who had had in addition the experience of 14 years' residence in Egypt and at El Tor upon which to draw. Any criticism, therefore, which may be directed against the amœbic part of this paper must be held to refer only to that one of us (J. G. W.).

Infantile diarrhœa, due, practically speaking, to Alexandria to the *B. dysenteriæ*, levies an appalling toll amongst Egyptian babies at the present day.

Amœbiasis, on the other hand, is a disease of protozoal origin, by reason of which it is much less infectious than bacillary disease, but immunity to it is less easily acquired and less complete.

It thus follows that the lower class Egyptian with his dirtier habits constantly exposes himself to both infections, but succumbs, as a rule, to the one only, unless his constitution has been previously strained by excessive hardship or disease. The better class Egyptian, on the other hand, thanks to his cleanlier habits, is usually exposed only to the bacillary infection, against which, owing to its far greater infectivity, precautionary measures are more difficult to apply.

The conclusion at which we arrived together—namely, that a diagnosis between the two great types of dysentery, amœbic and bacillary, can be made not only more rapidly but even with greater accuracy by simple direct microscopic examination of the stools than it can by cultural methods—has been strengthened by this further study and by a consideration of the results published by independent workers, particularly Bahr, Wenyon and O'Connor, Ledingham and Mackie, to mention only a few with whose work we are most familiar.

The results in particular of the investigations conducted by or under the ægis of the Medical Advisory Commission have established beyond all manner of doubt the contention which we, in 1915, were almost alone in upholding—namely, that bacillary dysentery has been, and is, the predominating and the more severe type of the disease in the whole of the Near Eastern area of operations.† To this conclusion we were led by investigations the object of which was to determine whether or not two diseases so distinct in their etiology and morbid anatomy would give rise in the dejecta to different types of cellular exudate, each peculiar to its own class—in other words, to determine whether a diagnosis could be made without identifying either the one or the other causal organism.

Importance of Early Diagnosis.

It is our purpose to show in this paper that—

1. Pure amœbic dysentery—i.e., uncomplicated by coexistent bacillary infection—gives rise to a characteristic exudate in the stools.

2. Bacillary dysentery, whether complicated by amœbic infection or not, also gives rise to a cellular exudate of specific character in the stools.

3. The finding of *Entamœba histolytica* in the midst of such a "bacillary" exudate indicates, not that the case is one of simple amœbic dysentery, but that a double infection is present, although all attempts to isolate dysentery bacilli may fail—as they do in many instances even when the case is one of "simple" bacillary dysentery.

4. In such cases of double infection, which are of more frequent occurrence than has been supposed, the diagnosis of the amœbic moiety can be made only by finding *E. histolytica*, for the reason that the exudate associated with the latter is comparatively so scanty and insignificant that it is masked by the overwhelming bacillary exudate. While the consequences of neglected amœbic infection may be disastrous, it is of still more immediate importance that bacillary dysentery should receive prompt recognition and appropriate serum treatment, for the following reasons:

(*a*) In fulminating cases, the time limit during which treatment is of any avail is much shorter in bacillary than it is in amœbic cases.

(*b*) In severe cases the patient rapidly becomes soaked with bacillary toxin, which results in severe degenerative changes occurring in myocardium, liver, and kidneys.

(*c*) The earlier a bacillary case is treated with *massive* doses of antiserum the more prompt and complete is recovery. While a delay of a day or two in instituting emetine treatment usually makes little difference to an amœbic case, a like delay in administering antitoxin to a bacillary patient may make all the difference between the abortion of the illness and months of invaliding and abdominal misery.

(*d*) Antidysentery serum is harmless to an amœbic patient; emetine, on the other hand, seems to us, as a result of our experience, to be a highly risky drug to administer to anyone whose myocardium is degenerated. As this does not occur in simple amœbic dysentery there is little risk in administering it in large doses to such cases, and even less to healthy carriers.‡

Pathology.

In order that the principle on which this cyto-diagnosis is based may be more fully understood it is necessary to give the briefest possible account of the pathology of the two diseases.

* From September, 1915, up to July, 1917, I (J. G. W.) was unable to obtain amœbic material in quantity sufficient for my purpose from European troops. All my cases of amœbic infection occurred in Egyptians of low class, among whom alone does the amœbic type of dysentery predominate. The explanation of this would appear to lie in their relative immunity to infection by the dysentery bacilli—a fact which is paralleled by their well-known immunity to organisms of the enterica group, which they have acquired through generations of existence in defiance of the most elementary rules of personal hygiene.

† We do not deny that there was a great deal of amœbic dysentery also in the summer of 1915, but not later.

‡ It must not be forgotten that *Entamœba coli* may coexist, as a harmless commensal, with *E. histolytica* or any of the dysentery bacilli.

By the term "dysentery" we understand a clinical condition the essential characteristic of which is the destruction of any part of the mucosa of the large intestine by one of the specific infections, amœbæ or dysentery bacilli; only those cases which showed evidence microscopically in the stools of such destruction in the form of mucosal sloughs and cellular exudate are included in the tables of our cases. Thus it is likely that many cases of early infection by dysentery bacilli which were recognised as such by the clinicians and treated immediately by them with massive doses of antiserum—with consequent abortion of the illness—figure in our returns simply as cases of enteritis. This was due to the fact that during the rush of the epidemic the pressure of work prevented the stools of all cases being examined until some days after the admission of the patient to hospital.

Pathology of Amœbic Dysentery.

Amœbic dysentery is due to infection by the *Entamœba histolytica*, and by it alone. No other amœba is capable of causing this disease, and it requires no help from secondarily infecting organisms of the bowel in the production of its lesions.

The life-history of this parasite is briefly as follows:—

The encysted form is swallowed with the food. Arrived in the small intestine, the digested juices there dissolve the capsule and set free the small daughter amœbæ, which make their way to the large intestine and thence down the lumina of the mucosal glands into the submucosa. There they grow into the adult or vegetative form, multiply by binary fission, and, in our opinion, give rise to a direct colliquative necrosis of the submucosa and muscularis mucosæ by means of a proteolytic ferment which they are able to secrete.

Direct communication with the lumen of the gut—i.e., ulceration—is only attained when either the solvent attacks the intercellular cement of the mucosa or the necrosis extends far enough to interfere with its blood-supply. According to which occurs first the mucosal cells are shed singly or *en bloc*, and an irregular ulcer with ragged, undermined edges is formed. The loss of substance is far greater in the submucosa than in the mucosa, as large vegetative amœbæ are found in the greatest numbers there; often sinuses lead from one ulcer to another under bridges of perfectly healthy mucosa.

As the amœbæ approach the surface of the mucous membrane again they divide more and more rapidly, and consequently become smaller and smaller until on the surface of the gut they assume the "*minuta*" form. Many of them now encyst, and they are passed out of the body in the stools as cysts or *E. minutæ*. It is worthy of note that the *E. minutæ* § produce no destruction of the tissues, and consequently no symptoms of dysentery; a fact which is distinctly in favour of the larger, or older, amœbæ producing a proteolytic ferment.

Adami was the first to point out that the reproductive and the higher functional activities of cells are mutually antagonistic; the more embryonic the cell the more readily does it multiply, and thus the more rapid is the growth of a neoplasm composed in greater part of cells of embryonic type. The smaller size of these amœbæ is due to their more rapid division, which has used up all their reserve of energy; consequently none is left for the development of that particular function which differentiates *E. histolytica* from all other amœbæ.

It has never been shown that the entamœbæ produce a diffusible toxin, or that they are capable of exerting any poisonous effect other than the proteolysis of cells in their immediate neighbourhood.

In consequence, the onset of the disease as a rule is insidious—it may be even unnoticed—and it is not until a comparatively large area of the gut is involved that the patient becomes at all acutely ill. It will thus be noted that the mucosal cells are in general healthy, apart from the areas of actual infection, while at autopsy the typical picture seen is one of areas of circumscribed necrosis, dotted like islands in the midst of healthy pink mucosa.

Now-a-days, when the emetine treatment is so universally adopted, cases where the bowel has undergone "gangrene en masse" are, fortunately, so rare as to warrant only passing notice. This condition is brought about by such an extensive amœbic invasion of the submucosa—and even the muscularis—with consequent thrombosis of blood-vessels, that the blood-supply of the bowel is cut off as effectually as by multiple thrombosis arising from any other cause.

The result is that the entire thickness of the bowel over a greater or lesser extent becomes gangrenous, and is passed as stringy black sloughs having a horribly fœtid odour. On very rare occasions it has even been impossible to remove the colon at autopsy, as all that remained to represent it was a slimy, stringy, black mass lying in a bath of corruption. Unless one had actually seen such a case it would be inconceivable that a man could survive long enough for his intestines and his peritoneum to attain such a state.

Sellards and Baetjer have shown that fatal cases of amœbic dysentery often terminate with a blood infection by members of the *Streptococcus fæcalis* group.

There is nothing astonishing in this if one considers how often the intestinal bacteria invade the body before death in different chronic and subacute diseases. The astonishing part is that it does not occur more frequently in amœbic dysentery—a disease in which there is such an extensive solution of continuity of the mucosal barrier. The point which we wish to emphasise is that in the vast majority of cases of non-fatal amœbic dysentery no such invasion of the blood stream can be demonstrated by hæmoculture.

This may be due to one or two causes acting together or separately.

In the first place, the liver may act as an efficient bacterial filter; in the second, the bowel itself is probably endowed, even when diseased, with a high immunity against just such organisms. Wright has shown that different parts of the body possess different degrees of resisting-power against pyococci. It seems reasonable to suppose that this local immunity acquired by countless minute inoculations with the bacteria-containing fluid in which the mucosa has been bathed since birth is sufficient to protect not only the body against invasion by these bacteria, but even the mucosa itself when the latter is partially destroyed by amœbæ.

In amœbic dysentery only when extensive areas are killed by interference with their blood supply does any secondary infection of the mucosa take place, just as a limb may become affected by wet gangrene after the blocking of its blood-supply. When, however, a pathogenic organism, such as a dysentery bacillus, which is a stranger to the mucosal barrier, and against which the mucosa is in consequence not immunised, arrives on the scene in sufficient numbers and virulence immediately its destructive properties come into play.

When sections are made through the edges of early amœbic ulcers, and more particularly when the unbroken amœbic nests in submucosa or liver are examined histologically, the salient point to strike the observer is the absence of the usual cellular infiltration characteristic of inflammatory reaction.

This is all the more striking when a comparison is made with the corresponding sections of early bacillary dysentery which is an inflammatory disease.

Again, the so-called amœbic liver "abscess," when sterile, is not an abscess at all. It is a primary colliquative necrosis of hepatic tissue, which is broken down and changed by a proteolytic enzyme from protein to albumoses and peptones; the patient's symptoms are due to the absorption of these poisons directly into the blood stream and can be reproduced experimentally by the intravenous injection of peptone.

To sum up: the pathology of histolytica amœbiasis is a primary degeneration due to the chemical digestion of the cells in the *immediate* neighbourhood only of the amœbic enzyme. It is not a primary inflammation with consequent degeneration; any inflammation which occurs is due to secondary microbic infection, is generally not severe, and takes place late in the disease.

The nature of the cellular exudate in the stools and in the uncontaminated liver "pus" is in entire accord with this basal fact of its pathology.

Pathology of Bacillary Dysentery.

Very different from the amœbic lesions are the changes which occur in bacillary dysentery. The infecting agents belong to a different kingdom.

The two diseases are so totally distinct that the only excuse for grouping them both under the heading of "Dysentery" is that of historical usage. In logic it were as reasonable to group cancer and tuberculosis of the intestine under the same heading because they may both give rise to what used to be called the "bloody flux."

Whatever may be the route of infection the essence of the pathology of bacillary dysentery is the intense diffuse catarrhal inflammation of the mucosa, which according to the severity of the attack may resolve or may go on to degenerative changes, such as coagulation-necrosis and sloughing. It is a primary inflammation of bacterial toxic origin, with all the cardinal signs of this condition—viz., "*rubor, tumor, calor, dolor et functio læsa.*"

The process is essentially one of diffuse destruction of the superficial layers of the mucosa, accompanied by œdema, large immigration of leucocytes, diapedesis of blood cells, and hæmorrhagic extravasation—such as is seen in all cases of acute bacterial inflammation of tissue. The immigration of polymorphs is an especially noteworthy feature. At autopsy the appearance of the intestine in any case of bacillary dysentery will vary with the intensity of the infection and the length of time the patient has survived it.

In the so-called fulminating ileo-colic dysentery, the entire surface of the lower part of the ileum and large intestine (*always excepting Peyer's patches and the appendix*) is seen to be the site of intense inflammatory change. No part escapes, as it does in amœbic dysentery; the bowel wall is œdematous, enormously thickened, deep plum-red in colour, and the mucosa may resemble the surface of a large granulating wound. A case of this intensity is more likely to be met with in patients who happen to be affected with renal disease of long standing, and death takes place from poisoning before the changes later to be described have had time to occur.

If the patient survives this stage, the superficial layers of the entire mucosa undergo coagulation-necrosis, become bile-stained, and are replaced by a green diphtheritic false membrane which may entirely hide the red granular layer underneath it. Here or there smaller or larger areas of this membrane may have sloughed, disclosing buds of granulation tissue of greater or lesser extent. Coincidently, small but numerous intramucosal hæmorrhagic extravasations take place.

If the patient again survives this stage small superficial ulcers, affecting particularly the summits of the valvulæ conniventes and mucosal folds, are formed. The minutely eroded, pinkish-grey "coraline" appearance of these ulcers, which run transversely across the bowel along the ridges of the mucosal folds of the colon, and are often present in large numbers, confers a very characteristic appearance. The sloughed mucosa is gradually replaced by well-formed and vascularised granulation tissue, and later by fibrous hyperplasia; if the patient recovers entirely the large intestine becomes shrunken into a thick-walled firm tube, with narrowing of its lumen and permanent damage to its functions.

Again, the ulcerative process may continue and spread, with the result that the gut wall may be honeycombed with myriads of small ulcers and so finally resemble a piece of red porous rubber.

§ They never contained red blood cells.

These ulcers, in spite of the fact that they may give rise to the so-called "worm-eaten condition," can easily be distinguished from the amœbic ulcers by the fact that they are more superficial, more regularly circular or oval, and have cleaner cut edges than the ragged undermined amœbic ulcers. Their depth will vary with the amount of fibrous change that has taken place in the bowel wall around them, which may be so great as to make them appear deeper than they really are. Whereas the thickened peritoneum may often constitute the base of a healed amœbic ulcer, it is extremely rare to find a bacillary ulcer penetrating deeper than the muscularis mucosæ. In all our experience we have yet to come across a case of perforation or peritonitis in pure bacillary dysentery.

Varying with the intensity of the infection, certain areas of the intestine are affected more than others.

Rogers states that the descending colon and rectum are more frequently involved than any other part. In our experience, on the other hand, the cæcum, first part of the ascending colon, and the flexures have been more profoundly affected than the other parts of the large gut.

When complete recovery has occurred after a less extensive infection than that first described the mucosa is often seen to be atrophied and stained a dirty slate-grey colour as a result of the diffuse intramucosal hæmorrhagic extravasation. In healed amœbic ulcers, on the other hand, this staining is seen only in and immediately around the cicatrices of the ulcers.

Concurrently with the local changes are those produced in heart, liver, kidneys, adrenals, and probably the nervous system, due to the absorption of the toxins formed in the gut by the dysentery bacilli.

In bacillary dysentery, as in diphtheria, the patient dies of intoxication. It may be definitely stated that as a general rule dysentery bacilli do not invade the blood stream, except during the stage of premonitory fever before diarrhœa begins. On one or two occasions certain observers have claimed to have isolated them by blood culture; but this invasion of the blood must be looked on as an accident in the course of the disease, and the occasions on which a general dysenteric bacillæmia takes place are so rare that the probability of its occurrence is negligible.

Just as the amœba is responsible for the ulcers of amœbic dysentery, so, in our opinion, is the dysentery bacillus alone accountable for the damage to gut and general system. We do not deny that secondary infection of the blood stream may and does take place occasionally in the course of both diseases, ‖ but we controvert the idea that any such infection is to be regarded as a regular concomitant.

It has been known for many years—in fact, since Todd first worked out the poison of Shiga's bacillus—that the "non-mannite-fermenting" type of dysentery bacillus produces a soluble toxin. Whether this toxin is really extracellular and diffusible from the first or whether it becomes so owing to a rapid breaking down of the bacillary bodies, and consequent liberation of endotoxin, is a matter of no moment. The fact remains that a filtrable toxin is easily demonstrated in cultures of this bacillus.

It has been the fashion to deny to the mannite-fermenting group of dysentery bacilli this attribute of being able to produce a diffusible toxin.

Everyone who has had experience of dysentery due to the mannite-fermenting type of bacilli must have been forced to the conclusion that a similar and equally virulent toxin was at work in these cases, as in those in which Shiga's bacillus was the morbific agent. The clinical symptoms are the same, and the degenerative changes in the organs seen at autopsy are indistinguishable from those seen in cases of death from Shiga's bacillus.

In May, 1913, one of us (J. G. W.), using a slight modification of the technique devised by the late Sidney Rowland to extract toxic nucleo-albumen from *B. pestis*, succeeded in obtaining from different mannite-fermenting strains of *B. dysenteriæ* a toxin which killed rabbits in doses of 0·01 g., in the same way as did the living bacilli. This toxin was afterwards used by one of us with very good results in the manufacture of antidysenteric serum from horses. This point is mentioned here to prove that whatever the type of infecting dysentery bacilli similar toxic changes occur in all.

Diagnosis Based on the Cytological Picture of the Stools.

It will thus be seen that it is easy to diagnose between the two great types of dysentery when they reach the post-mortem table, so definite and peculiar to each is its morbid anatomy. We were led, therefore, to hope that on *a priori* grounds the differences in the nature of the exudate and desquamated cells found in the stools of each variety would be sufficiently constant and characteristic to constitute a basis for differential diagnosis at once reliable and rapid.

Our investigations have led us to the conclusion that it is possible to arrive at such a diagnosis, based entirely on the cytological picture peculiar to each type of the disease.

The character of any given stool will depend upon the particular stage of the disease at which that stool has been evacuated. It will depend to a much less extent upon the nature of the food which the patient has taken, in that in the vast majority of cases both types of patients—amœbic and bacillary—will have been treated on similar dietetic lines; further, our examination is restricted to any sloughs, bloody mucus, &c., which may be mixed with the fæcal matter.

‖ Embolic suppurative nephritis (renal pyæmia) was a not uncommon termination of fatal cases of bacillary dysentery among pilgrims at El Tor. A form of *B. coli* was always isolated from the emboli—never a dysentery bacillus.

Cyto-diagnosis in Amœbic Dysentery.

If some of the bloody mucus from a case of "simple" amœbic dysentery be examined microscopically (under the ⅓" and ⅙" objectives) the first thing to strike the observer will be the scantiness of the cellular exudate, though red-blood corpuscles may be very abundant. This point has particularly impressed us when examining material taken from the surface of an ulcer at autopsy.

Another salient characteristic is the preponderance of mononuclear cells over polymorphonuclears, a feature which contrasts sharply with the picture seen in bacillary dysentery. (Compare Figs. 1 to 9 with 12 to 15.)

Next to the scantiness of the cellular exudate the most noteworthy element in the film is the picture presented by the actual cell components—most of them look half-digested, and have a "mouse-eaten" appearance (Figs. 10 and 11). The process evidently begins at the periphery, and the nucleus is the last to be involved. In many cases groups of nuclei appear to become agglutinated by means of their semi-digested surrounding cytoplasm.

The process is apparently a slow one, the cytoplasm being gradually digested and dissolved and the unchanged nucleus being left bare or clothed merely in a narrow strip of ragged cytoplasm. Fig. 5 shows this very clearly; the stool was obtained 24 hours before the death of the patient, an Egyptian, from acute amœbic dysentery. At autopsy the entire large intestine was found to be studded from end to end with amœbic ulcers of all shapes and sizes; non-agglutinating vibrios were obtained in culture, but no dysentery bacilli could be isolated. Of far greater value in demonstrating the absence of bacillary infection in this case was the healthy pink colour of the mucosa between the ulcerated areas. (The diagnosis was made at first from the nature of the cellular exudate alone, though amœbæ were found later.)

We have recently had occasion to examine one of the very advanced cases of gangrenous amœbic dysentery described earlier in this paper. (Figs. 7 and 11.)

Curiously enough, this patient (an Egyptian) also harboured non-agglutinating vibrios in his intestines, but at autopsy there was no evidence of cholera and no vibrios could be isolated from the small intestine. Here again it was possible to make an ante-mortem diagnosis of the condition of the large bowel by means of the stringy black sloughs, the appalling fœtor of the stool, and the fact that microscopically hardly any polymorphonuclear cells were seen—nothing but amorphous débris apparently more or less completely digested, and a few nuclei from which all the cytoplasm had been stripped. Many of the nuclei, even, were semi-digested and degenerated; but of all the structures involved they were evidently the most resistant to the degenerative force in action.

In our opinion, this type of degenerative change can be explained only on the supposition that in amœbic dysentery no toxin is at work in the organism at all comparable to that produced by the dysentery bacilli.

As will be shown later, the cells in a bacillary stool give evidence of being poisoned *en bloc* and undergo rapidly the changes characteristic of toxic necrosis and autolysis, the nucleus being among the first parts of the cell to be affected.

Further, the fatty changes to be described as occurring invariably in cells composing the bacillary exudate are never seen to anything like the same extent in amœbic stools, when examined fresh in Sudan III. solution; all that one sees are isolated "lumps" or dots of fat, suggesting that the cells had previously ingested some fatty material rather than that it was of intrinsic cytoplastic origin.

Consequently, all the evidence goes to prove, in our opinion, that every morbid change seen in amœbic dysentery can be explained on the hypothesis of proteolytic digestion of living tissue by the amœbæ.

To sum up the characteristics of a "simple" amœbic stool:—

1. Scantiness of cellular exudate, especially the polymorphonuclear element.
2. Preponderance of mononuclears over polymorphonuclears.
3. Evidence of proteolytic digestion of the cells, beginning at the periphery and affecting the nucleus last.
4. Absence of all phenomena characteristic of inflammatory reaction, toxic necrosis and consequent autolysis.

Cyto-diagnosis in Bacillary Dysentery.

A very different picture is seen on examining microscopically the blood-stained muco-pus from a case of bacillary dysentery, whether in the acute, subacute, or chronic stage.

Here the first feature to strike the observer is the abundance of cellular exudate, composed largely of polymorphonuclear leucocytes. Desquamated and degenerated mucosal cells are also common, but the polymorphonuclear neutrophiles predominate. Blood may or may not be present according to the stage of the disease, whereas it is hardly ever absent from the stools of amœbic dysentery.

The polymorphonuclear neutrophiles show the characteristic changes consequent on toxic necrosis more than any other leucocytes, the eosinophiles less and the lymphocytes least of all. The cell is evidently poisoned *en masse*, and whether the further changes about to be described are directly caused by the toxin or are merely autolytic phenomena resulting from the death of the cell is of no moment. The important point for our purpose is that the process is obviously different from that which takes place in the cells of an amœbic exudate.*

* The nuclei seem to be specially sensitive to the action of this toxin, as the changes described, particularly fragmentation and agglutination of small chromatin masses round the nuclear membrane, and "ringing" of the nuclei, have not been observed by us in similarly prepared films of pus obtained from other sources—e.g., boils—or at least to nothing like the same degree.

A type of cell which is always present, often in large numbers, and which seems to be associated more frequently with bacillary (especially cases which are improving clinically) than with amœbic dysentery somewhat resembles a free resting amœba, and it is important not to mistake it for one. (Figs. 17 to 19.)

There is, in our opinion, little doubt that this mistake has been often made, and that as a result the numbers returned as amœbic dysentery in 1915 were unwarrantably high. Particularly do we consider these remarks applicable to cases of so-called "running amœbic dysentery," often accompanied by a rise of temperature, which responded beneficially to serum treatment. These cells may have also given rise to the diagnosis "post-amœbic colitis."

As these cells are larger than a polymorphonuclear leucocyte, and as the changes in them are much the same as those occurring in other leucocytes, it will be convenient to describe them as a type.

They are very large mononuclears, 15 to 30μ in diameter, with basophilic non-granular cytoplasm, and are probably macrophages of endothelial origin. They are possibly identical with the cells described by Jurgens, Dutcher, and Bahr in the stools of bacillary dysentery some years ago.

The cytoplasm, if not too degenerated, contains numerous vacuoles in which there may be entire polymorphonuclear leucocytes, lymphocytes, red cells, or their fragments, and bacteria, more or less digested. Curiously enough, the ingested leucocytes are much less degenerated than their hosts. By Achard's method of eosin staining these large cells can usually be demonstrated to be dead. In others, as a result presumably of more advanced degeneration, the cytoplasm, instead of being granular and vacuolated, is swollen, hyaline, transparent, and apparently in a state of hydropic degeneration; it stains very poorly or not at all with iron-alum-hæmatoxylin.

The degenerative process begins early in the cytoplasm, which first becomes granular, then vacuolated, and finally hyaline. It may show dots or granules, which are greenish when examined in the fresh state and intensely black when stained with iron-hæmatoxylin.

These are probably fragments extruded from the pyknotic nuclei. In fresh specimens these dots are frequently seen to exhibit distinct Brownian movements and are apt to be mistaken for bacteria or other debris which the cell has ingested. Coincidently with the cytoplasm the nuclei begin to show very definite karyorrhectic and karyolytic changes, both in fresh and stained specimens.

The nucleus first becomes swollen, then granular; finally its centre becomes transparent and the chromatin is aggregated under the nuclear membrane, leaving the centre bare. (In other cases pyknosis may be the only change, as seen in the stained specimens.) The nucleus then bursts, and the granules resulting from this action appear as a scattered line of irregular dots—green in the fresh and black in the stained specimens—which are apparently caught in the periplast, all that remains of the cytoplasm.

Later these dots disappear, and the cell is represented only by the periplast, which is usually regularly circular. (Fig. 15.) These cells, as their suggested origin implies, possess certain feebie powers of amœboid movement, a characteristic which is liable to entrap the unwary.

Fig. 18 shows one of these macrophages (fixed and stained by the Schaudinn-Heidenhain method), which suggests that it has been caught in the act of throwing out a pseudopodium.

These endothelial macrophages resemble resting or dead amœbæ most closely when they show the aggregation of chromatin granules scattered around the periphery of the nucleus; then one sees a large round cell, vacuolated and containing ingested cells, with a nucleus which exhibits the "ringing" so characteristic of the nucleus of amœbæ. The fact, however, that the nucleus in a degenerating amœba is the last structure to disappear may be of some guidance in differentiation.

Similar degenerative changes to those mentioned occur in the leucocytes and also to a less extent in the mucosal cells.

Fatty degeneration is always present in the cells composing the exudate in bacillary dysentery. In the very early stages this change is very slight and the intracorpuscular globules present in leucocytes and desquamated mucosal cells appear as dust-like red granules when examined under the immersion lens, and a Sudan III. in alcoholic solution is run in under the coverslip.

Later these globules become larger and larger, until in the advanced cases the greater part of the cell body is filled with them. In some we have even seen the leucocytes distended with large red globules crammed together inside the periplast like potatoes in a sack. This condition is best seen in the liquid green pea-soup type of stool, without obvious muco-pus or blood, characteristic of advanced subacute or chronic bacillary dysentery. (Fig. 10.)

Another point to strike the observer in cases of *acute* bacillary dysentery is the extraordinarily small number of bacteria present; the film, in fact, looks more as though it had been taken from the pus of a streptococcal abscess than from a stool. (Contrast Figs. 3 and 12.)

To sum up the characteristics of a bacillary stool:—

1. Abundance of cellular exudate, mostly polymorphonuclear.
2. Preponderance of polymorphonuclears over mononuclears.
3. Evidence of toxic necrosis of cells, the degenerative changes occurring early in all parts of the cell, including the nucleus. This degeneration may go so far as to leave only the circular periplast of the cytoplasm, thus constituting the "ghost-cell," the presence of which in any quantity is very typical of bacillary infection.
4. Evidence of phenomena characteristic of intense inflammatory reaction to microbic infection.

Results of Cultural and Cyto-diagnostic Methods.

The cultural method of diagnosing bacillary dysentery is of value only when positive results are obtained.

It is, at best, a difficult operation—how difficult in all but the early acute cases only those who have had years of experience at this work fully appreciate. It is slow, laborious, uses up an enormous amount of glass-ware and medium, and necessitates a fully equipped laboratory with a well-trained staff. All these difficulties are multiplied under active service conditions, and we have no hesitation in saying that the experience of this campaign has shown it to be a complete failure, alike for statistical and clinical purposes, in that results reach the clinician too late to be of any guidance to him in his treatment.

We contend that our method of cyto-diagnosis is not only much more rapid and less laborious, but also that statistics based on it give a much truer idea of the incidence of bacillary infection than do any that are based on the isolation of dysentery bacilli—at least, under active service conditions.

A study of the various reports issued periodically by the field laboratories shows how far their return of positive findings falls below the real figure of bacillary cases. In one instance this represented only 7 per cent. of "blood and mucus" cases in which an expert protozoologist could find no amœbæ. The bacteriologist in charge at the time was a man of exceptional ability and long experience in this particular subject.

Appended are the figures of "cases of dysentery and suspected dysentery" for all the laboratories of Egypt—including those at the various bases under the direction of experts working under optimum conditions—during the first quarter of 1917.

1. Total number of cases examined	4037
2. Number showing blood and mucus	1127
3. Number of cases microscopically examined	3818
4. Dysenteric amœbic findings	300
5. Number of cases bacteriologically examined	3094
6. Dysenteric bacilli, typical and atypical, isolated	475

No. 6 gives 15·3 per cent. of No. 5.

Even if the whole of No. 4 be subtracted from No. 2 the percentage of No. 6 to No. 2 is only 57·4. Even this is too high a figure, as cysts are included in No. 4, and such stools usually do not contain blood and mucus. Further, no account is taken of mixed infections.

Mutability of Organisms.

Again, the cyto-diagnostic method has the further advantage of obviating a difficulty which recent experience has shown to beset the cultural method of diagnosing bacillary dysentery.

We refer to the doubt often present in the bacteriologist's mind as to whether a given organism which he has isolated is to be regarded or not as the actual cause of the disease. There is no doubt that the group of bacilli capable of causing dysenteric infection is a very large one, with much more elastic bounds than it has hitherto been customary to allot to it. This applies particularly to the so-called "mannite-fermenting group," the membership of which has of late years been expanded to admit a host of new candidates, all of which show a certain relationship in their serological and biochemical reactions to the classical Flexner-Y types.

The bacteriological work of the present campaign, while adding to our knowledge, has at the same time added vastly to our confusion in this respect.

Almost weekly a new type of bacillus—non-motile, Gram-negative, anaerogenic, non-lactose-fermenting—has turned up on our plates, each new type showing in infinite gradation affinities with, and divergence from, the classical in its bearing towards recognised agglutinating sera and fermentation of sugars. For instance, to take only two examples from opposite ends of the scale, we have on several occasions isolated a bacillus having all the bio-chemical and cultural characters of a typical *B. Shiga*, but showing no agglutination whatever with any Shiga-agglutinating serum at our disposal.

One such strain—isolated from one of us after a mild attack of dysentery—was as virulent when tested in rabbits as any type of *B. Shiga* in our possession.

Another bacillus was agglutinated up to 1:2000 by Y-serum from the Lister Institute, though not by normal serum, Shiga or typhoid serum in low dilution; yet this strain—the purity of which was tested by replating—fermented every sugar on which we tried it. It produced permanent acidity, but no clot, in milk.

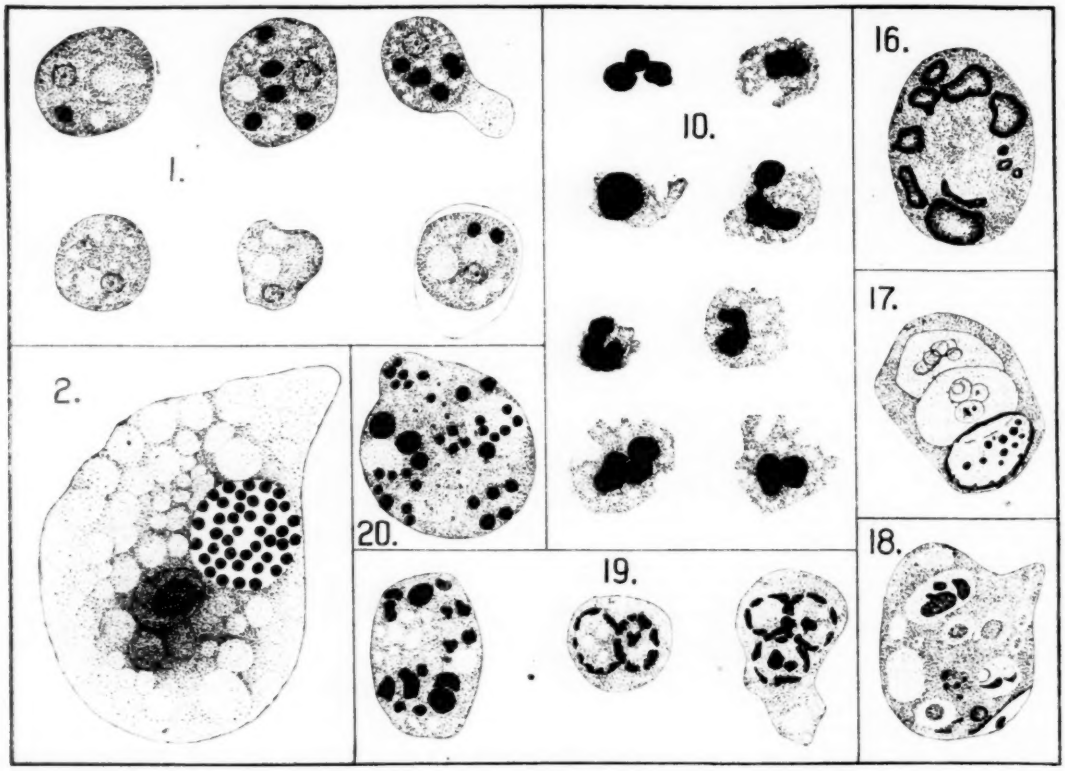
We may add here that the results of bacteriological investigation of a large series of cases of bacillary dysentery in the Central Laboratory, Alexandria, fully confirm such statements.

With all diffidence we hazard the suggestion that all pathogenic bacteria having their habitat in the intestine are actually or potentially mutable.** Apart from the facts bearing on mutability of the organisms of the enterica and dysentery families, we feel tempted in this connexion to quote others of a similar nature, relating to this phenomenon.

Thus, Bordet has shown that his *B. pertussis* could be changed by repeated subculture into an organism having entirely distinct cultural and agglutinative reactions.

** *Cæteris paribus*, the more nearly the infecting bacillus approaches the classical type, the more severe is the course of the disease, and the more prompt the response to serum; and vice versa.

FÆCAL FILMS FROM CASES OF ACUTE AMŒBIC (1–11) AND BACILLARY DYSENTERY (12–20).



DESCRIPTIONS OF ILLUSTRATIONS.

1. *Amœbic Dysentery.*

FIG. 1.—Types of *Entamœba histolytica*. Stained Schaudinn-Heidenhain-Wenyon method. Zeiss 1/12, Oc. II.

FIG. 2.—*Entamœba coli* with degenerated and vacuolated cytoplasm and ingested bodies, parasites. Note the unaffected appearance of the nucleus. Drawn in black and white, but stained as follows: Schaudinn-Heidenhain (Wenyon's modification): Decolourised completely in acid alcohol —> ammonia alcohol, and 50 per cent. spirit, —> counterstained 12 hours in Borrel's blue 1 c.cm., Gauducheau's stain 4 c.cm., dist. water 5 c.cm. Washed in 50 per cent. alcohol, to which a little 1 : 5000 acetic acid was added —> 0·5 per cent. eosin in alcohol —> absolute alcohol —> differentiated in oil of cloves (controlled under microscope) —> xylol —> Canada balsam.

By this method nuclear chromatin is stained blue-black, neutrophile cytoplasm pink, red blood cells crimson. The advantage of this method over the original Schaudinn-Heidenhain is that the cells inclusions can be differentiated from fragments of chromatin, and the nuclei of leucocytes, from which the cytoplasm has been stripped, from red blood cells. (This method is referred to hereafter as "W" stain.)

From a case of amœbic dysentery. Zeiss 1/12, Oc. VIII.

FIG. 3 (original in colours, but reproduced in black and white).—Fæcal film from a case of acute amœbic dysentery. "W" stain. Zeiss 1/12, Oc. II. Note the preponderance of mononuclears, the mouse-eaten appearance of the cytoplasm, the stripping of the unaffected nuclei. An amœba is seen, showing the characteristic nucleus.

FIG. 4 (original in colours).—Fæcal film from a case of acute amœbic dysentery. Modified "W" stain. Zeiss 1/12, Oc. II. An amœba containing red blood cells is seen, but the nucleus is not so obvious by this stain, as the iron hæmatoxylin was omitted.

FIG. 5.—Fæcal film from a fatal case of amœbic dysentery. This man died with symptoms of cholera, and vibrios (non-agglutinating) were present in the stools. Autopsy revealed multiform amœbic ulcers, thickly studding the large intestine from cæcum to rectum. The peritoneum was full of ascitic fluid; the spleen was hard and enormously enlarged; the liver in an advanced state of cirrhosis—a case of "Egyptian splenomegaly" (Ferguson).

FIG. 6 (original in colours).—Fresh fæcal film from a case of acute amœbic dysentery. Note the scantiness of cellular exudate. Zeiss 1/12, Oc. II.

FIG. 7.—Film from surface of colon—Schaudinn-Heidenhain-Wenyon stain. This was taken at autopsy from a case of advanced gangrenous amœbic dysentery such as is described in the text. Zeiss 1/12, Oc. II. Note the digested appearance of the cells.

FIGS. 8 and 9.—Fæcal films from fatal cases of amœbic dysentery—Schaudinn-Heidenhain-Wenyon stain. Zeiss 1/12, Comp. Oc. VI. Compare the macrophage containing a lymphocyte and red blood cells in Fig. 9 with the amœba in Fig. 8. Note that the nuclei of leucocytes and mucosal cells are unaffected, while the cytoplasm has suffered severely. In some instances it has disappeared, leaving the nucleus bare.

FIG. 10.—Leucocytes from fæcal films of amœbic dysentery, to show digestion of cytoplasm. Schaudinn-Heidenhain-Wenyon stain. Compare the nuclei with those seen in bacillary films.

FIG. 11.—A single field from the intestinal contents of the case of gangrenous amœbic dysentery mentioned above, showing a lymphocyte, a mucosal cell, a polymorphonuclear leucocyte, and a red blood cell. Schaudinn-Heidenhain-Wenyon stain. Zeiss 1/12, Comp. Oc. VIII.

II. *Bacillary Dysentery.*

FIG. 12 (original in colours).—Fæcal film from a case of acute bacillary dysentery. "W" stain. Zeiss 1/12, Oc. II. *B. Shiga* isolated. Compare with Fig. 3.

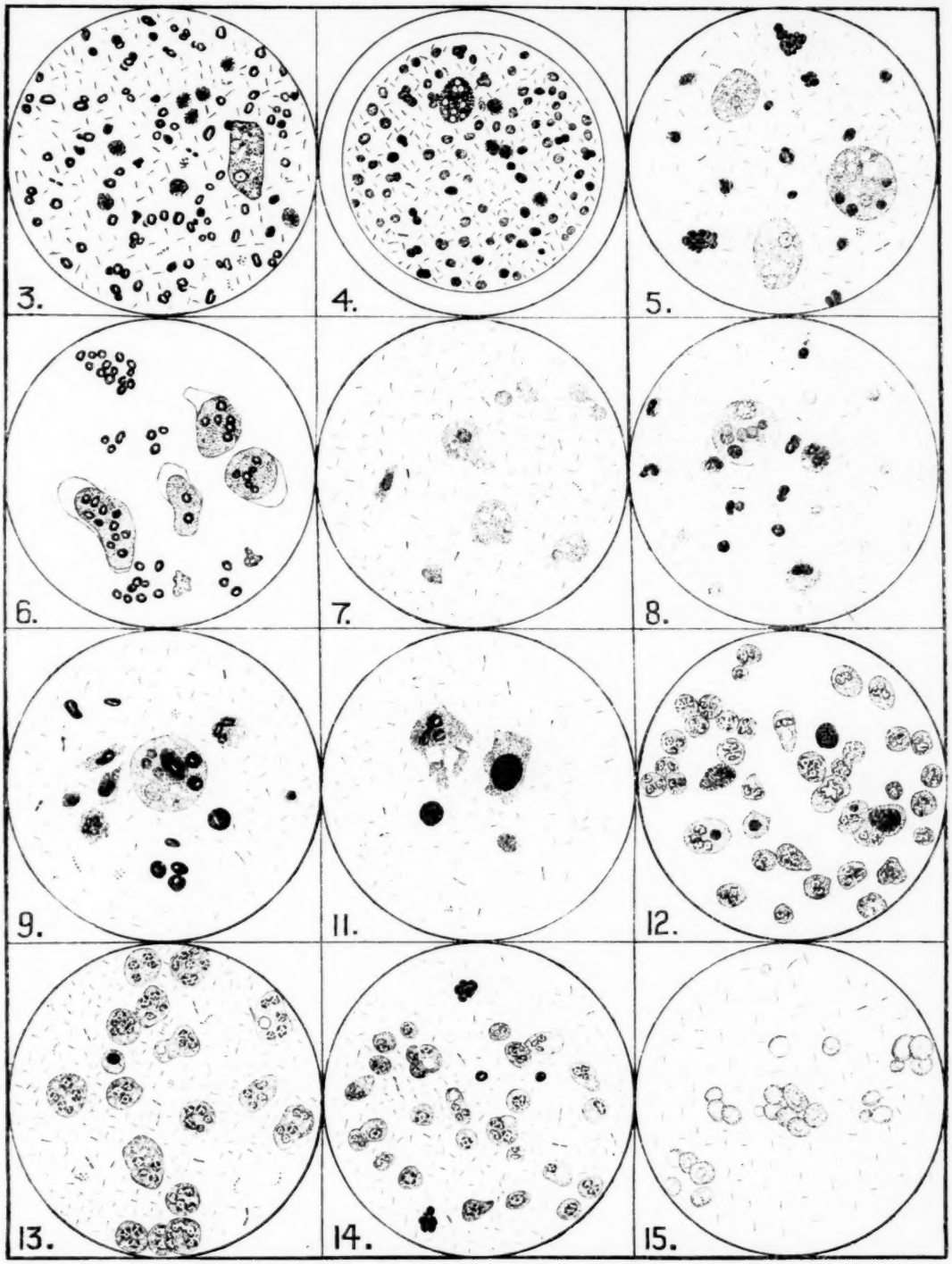
FIG. 13.—Fæcal film from a case of acute bacillary dysentery. "W" stain. Zeiss 1/12, Oc. II. *B. dysenteriæ* mannite-fermenting type isolated. Drawn in black and white. The cell at 1 o'clock contains an ingested red blood corpuscle.

FIG. 14.—Fæcal film from a case of acute bacillary dysentery of about 1 week's duration (Schaudinn-Heidenhain-Wenyon stain, Spencer 1/12, Oc. II. *B. dysenteriæ* mannite-fermenting type isolated). Note the fragmentation of the nuclei and the regular outline of the cytoplasm.

FIG. 15.—Fæcal film fresh preparation in saline from stools of subacute bacillary dysentery unstained. Spencer 1/12, Oc. II. (*B. dysenteriæ* mannite-fermenting type isolated.) To show degenerative changes in leucocytes and formation of "ghost cells."

FIG. 16.—Polymorphonuclear neutrophile probably hydropic, from fæcal film of bacillary dysentery, "W" stain, drawn in black and white. Zeiss 1/12, Oc.V. To show fragmentation and centrifugal tendency of nuclear chromatin.

FIG. 17.—Macrophage of endothelial origin from a case of subacute bacillary dysentery (*B. dysenteriæ* mannite-fermenting type, probably *B. Tor No. 1*, isolated). Schaudinn-Heidenhain-Wenyon stain. Reichert 1/12, Oc. XII. The nucleus shows early karyolysis, two polymorphonuclear leucocytes ingested.



DESCRIPTIONS OF ILLUSTRATIONS (*continued*).

FIG. 18.—Shows a similar cell with much more advanced degenerative changes from another case (technique as in Fig. 17). The cytoplasm is swollen, probably hydropic, and shows a projection resembling a pseudopodium towards 1 o'clock. Inclusion forms are represented by a polymorphonuclear leucocyte, a lymphocyte, red blood cells, and their fragments. The nucleus is not obvious, but is probably represented by a line of pyknotic chromatin granules near the periphery of the cell from 4 to 7 o'clock. There is no reason to suppose that any of the changes are artificial or extra-corporeal. (Flexner's bacillus isolated in almost pure culture from the stool.)

FIG. 19.—Leucocytes from a case of bacillary dysentery (Schaudinn-Heidenhain-Wenyon stain. Zeiss 1.12, Compens. Oc. VIII.). The largest cell is probably a macrophage with pyknotic nucleus, the smallest probably a polymorphonuclear leucocyte, and the cell of irregular contour possibly a macrophage, showing the nuclear stigmata peculiar to degeneration produced by toxins of bacillary dysentery.

FIG. 20.—A leucocyte from a fresh preparation stained with Sudan III. from a bacillary stool. To show fatty degeneration of cytoplasm (drawn in black and white). Zeiss 1.12, Oc. VIII.

MacConkey—most suggestive of all—showed that an old stock culture of *B. diphtheriæ*, which had completely lost its toxigenicity, regained this property concurrently with an epidemic outbreak of diphtheria in the same district as that from which this strain had been originally isolated.

One of us has noticed a similar variability in the virulence of stock dysentery cultures. Again, one of us (C. H. S.) has recently recorded—at the end of a true enterica epidemic—a small epidemic of a mild enterica-like disease, in which *B. fæcalis alkaligenes* was constantly present in the blood stream.

Crendiropoulo has drawn attention to the constancy with which non-agglutinating vibrios replace the agglutinating in the stools of cholera contacts towards the end of an epidemic, to disappear completely a certain time after the epidemic has ceased. He has also adduced a mass of evidence to show how vibrios may lose or acquire certain of their characteristics, such as agglutination, hæmolysis, and the production of cholera-red.

All the above, while perhaps not strictly relevant to the subject matter of our paper, appears to us as worthy of consideration, and as justification for our plea for greater latitude of judgment in the solution of problems presented by epidemic disease in general and epidemic dysentery in particular.

Our thanks are due to Captain Maguire, R.A.M.C., and to Corporal E. G. Davis, R.A.M.C., for the very accurate drawings from the microscope with which this article is illustrated (we are much indebted to them for the time and trouble which they have devoted to this work); to the clinicians in charge of the dysentery wards, especially Captain Fisher, R.A.M.C., and Captain Donald, R.A.M.C., for their most cordial coöperation; and, above all, to Major A. R. Ferguson, R.A.M.C., Officer Commanding the Central Laboratory, Alexandria. We have to thank him, not only for correcting this paper and for his unfailing help in any difficulty, but also for many valuable suggestions and the generosity with which he placed the knowledge gained during many years' experience of dysentery in Egypt at our entire disposal. We also wish particularly to thank Captain P. H. Bahr, D.S.O., R.A.M.C., who has had considerable experience of dysentery in the field, for criticisms and suggestions from his own work on the same subject.

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In the Field, Sinai, 1917.

NOTES ON RECENT EPIDEMIC OF SMALL-POX IN THE FIELD.

BY G. H. MEAD, M.R.C.S., L.R.C.P. LOND.,
LIEUTENANT, R.A.M.C. (T.C.)

HAVING recently had charge of a small-pox camp and the opportunity of seeing some 25 to 30 cases with the sole charge of 15 from the initial symptoms up to the termination of the case, I have thought that the following notes and observations might prove of interest to fellow-practitioners at home, where hitherto small-pox has been of rare occurrence, though owing to the laxity with which vaccination is now enforced an epidemic, I fear, may break out at any time in the near future.

Small-pox commenced to show itself among the troops in this theatre of war towards the end of October, 1917. I think there is no doubt that the infection was conveyed by the Arabs, among whom the disease is more or less endemic. The Arabs were in the habit of frequenting the camps for the purpose of selling their produce.

The fact that the disease was early got under control was, I think, entirely due to the following measures that were taken: 1. Vaccination or revaccination of the personnel of all troops in the infected area who had not been vaccinated within the past five years. 2. The early recognition of the first cases. 3. The rigid isolation of suspected cases, together with the isolation of all contacts. 4. The segregation of such troops from which definite cases had been removed. 5. The exclusion of Arabs from the camps.

Illustrative Cases.

Of the cases that came under my care the following seem to me to be of special interest as demonstrating the different types one meets with. These vary from the virulent confluent form in the unprotected or susceptible person at the commencement of the epidemic to the mild or modified form in the well protected person ending in the abortive type at the end of the epidemic as the disease loses its virulency and flickers out.

CASE 1. *Confluent variola.* (See Chart.)—Driver R., aged 43. Vaccinated in infancy, four large scars, never been revaccinated. Reported sick on Nov. 21st, 1917, with pains in head and vomiting. Was admitted into field ambulance; preliminary diagnosis of "gastritis" made. On the 23rd he was transferred to isolation camp with papular eruption on face, trunk, and extremities. Eruption on face very profuse; considerable inflammatory œdema. Likewise eruption of papules on back was very profuse and accompanied by intense erythema and œdema. By the 26th (fifth day of disease) eruption generally had reached vesicular stage; vesicles of characteristic loculated and umbilicated appearance. On face and back eruption had become confluent; on former œdema was so intense that patient could only with difficulty open his eyes.

Dysphagia was also a troublesome symptom, causing much distress to patient, and requiring great patience of nursing orderlies in feeding. He commenced to suffer from diarrhœa.

By Dec. 1st (tenth day) condition was hopeless and patient presented a truly terrible appearance. His entire features had become obliterated. He was unable to open his eyes at all and the skin of the face was literally floating on a sea of pus. Similar condition on the back, where large areas of skin sloughed off. Patient lingered on until the 4th (fourteenth day of disease), when death released him.

CASE 4. *Discrete variola.* (See Chart.)—Driver D., aged 33. Vaccinated in infancy, one large scar. Revaccinated 1915, unsuccessfully, no scar. Reported sick on Nov. 26th, 1917, with fever, headache, and backache; admitted to field ambulance and isolated. On the 28th was transferred to small-pox camp with papular eruption on face, trunk, and extremities; eruption fairly profuse on face, very profuse on back. By Dec. 1st (fifth day of disease) eruption was generally vesicular, profuse, but quite discrete, except for peculiar little clusters of confluent vesicles along each tendo Achillis. By eighth day pustulation was fairly complete, pustules on face commencing to collapse and desiccate. Subsequent course uneventful, desiccation proceeding satisfactorily and followed by branny desquamation at site of pustules. Some pitting noticeable on face.

CASE 7. *Modified variola.*—Private G., aged 33. Vaccinated in infancy, two good scars. Revaccinated May 16th, 1917, two good scars. Reported sick Dec. 2nd, 1917, with headache, dizziness and backache. Was admitted to isolation camp with scattered vesicular eruption on face, trunk, and extremities. Eruption extremely slight, vesicles having very poorly marked inflammatory areolæ around them, but showed definite loculation and umbilication; few scattered papules on palate. Subsequent course very mild. Pustulation was very slight and unaccompanied by any constitutional upset, the papules rapidly collapsing and desiccating.

CASE 9. *Confluent variola.* (See Chart.)—Pte. D., aged 28. Was vaccinated in infancy, arm showing four plain scars. Was isolated and successfully revaccinated as a contact of small-pox on Nov. 26th, 1917, and kept under observation. On Dec. 4th he complained of general malaise. On the 5th he was removed to small-pox camp with papular eruption on face, trunk, and extremities; considerable erythema and commencing œdema of face. By the 6th eruption was very profuse; papules very thickly set on face and œdema increasing. Back also profusely covered with papules. Papules plentiful in mouth, on palate, tongue, and lips. By the 7th œdema of face was intense; papules were becoming vesicular and confluent. Pustulation was severe and accompanied by considerable inflammatory reaction around pustules. By the 12th (eighth day of eruption) pustulation was pretty general, but œdema about face appeared to be subsiding and general condition markedly improved. Subsequently desiccation proceeded satisfactorily, followed by prolific desquamation of cutis in large pieces. Pitting was considerable.

CASE 11. *Hæmorrhagic confluent small-pox.*—Pte. G., aged 19. Unvaccinated. Reported sick on Dec. 15th, 1917, with pains in head and back. Admitted to small-pox camp on 19th with papular eruption on forehead, trunk, and extremities; papules most numerous on back. By the 21st (sixth day) eruption was very profuse; considerable œdema of face; papules were vesiculating and tending to become confluent. By the 24th (ninth day) pustulation was general and accompanied by hæmorrhage into pocks. Subsequently patient became toxæmic and at times violently delirious and died on the 29th (fourteenth day).

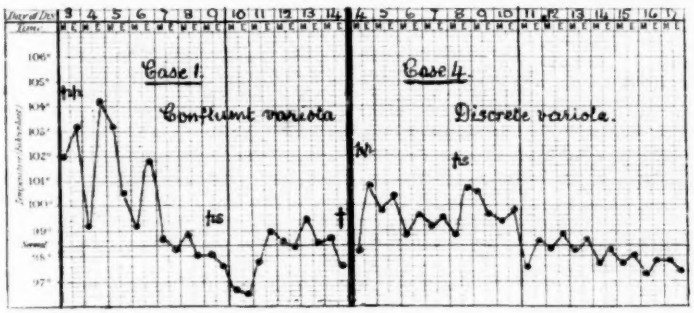
CASE 12. *Discrete variola with initial rash.*—Pte. R., aged 30. Vaccinated in infancy, four large scars. Revaccinated in 1914, no scars visible. Reported sick on Dec. 24th, 1917, with shivering, headache, and backache. On 25th petechial purpuric eruption appeared on lower half of abdomen, extending on to thighs. On the 27th (third day) papular eruption appeared on face, arms, thorax, back, and legs. By the 29th vesiculation was general, initial rash fading. Vesicles remained quite discrete. Pustulation was mild and subsequent course uneventful.

CASE 15. *Modified abortive variola.* (See Chart).—Driver O., aged 18. Vaccinated in infancy, four plain scars. Revaccinated Jan. 6th, 1918, two plain scars. Reported sick on the 9th, complaining of headache and diarrhœa. Was admitted to small-pox camp on the 13th with few scattered papules on face, arms, and back. Subsequently only a few papules matured, remainder aborting without reaching vesicular stage. On eighth day T. rose and pain in left shoulder; swelling, heat, and tenderness in region of left acromio-clavicular joint. The joint was wrapped in wool and immobilised by binding arm to body and aspirin administered. The trouble rapidly subsided; subsequent course uneventful.

Comments.

In Cases 1 and 11 the early appearance of inflammatory œdema of the face will be noted. This, in my opinion, is an important and grave sign. It is important as an early sign and aid to diagnosis, and is often evident when the papules are rather more palpable than visible. It is also a sign of grave prognosis, and when well marked points to a severe attack.

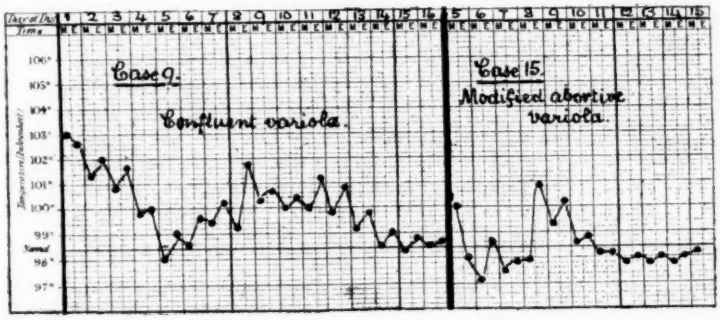
Case 9 is also of special interest in demonstrating that in this case at any rate revaccination did neither abort nor apparently modify the attack. The patient must have been incubating the disease when he was revaccinated, and the revaccination had apparently little or no influence upon the course of the disease. From this case alone one would gather that to prevent or abort the attack one must be revaccinated either before or immediately after exposure to infection.



Case 7 might be taken legitimately, I think, as an attack of variola modified, and consequently rendered mild, by the successful revaccination 11 months previously.

Case 11 is of special interest as indicating the severity of an attack in an unprotected person, also on account of the complication of hæmorrhage. Hæmorrhagic small-pox occurs in two forms. In the one, the variety commonly known as black small-pox or purpura variolosa, the constitutional symptoms are intense. Punctiform hæmorrhages occur under the skin and there may be also hæmorrhages from the mucous membranes in the first few days of the illness, and death may supervene before the papules are properly developed. In the other type, that this case exemplifies, the case progresses as a severe one and the hæmorrhages occur in the vesicular or pustular stage.

Case 12 is of a peculiar interest on account of the prodromal or initial rash. This rash, being of a purpuric character, might have indicated a severe hæmorrhagic type but for the fact that the constitutional disturbance was slight and everything pointed to the case being a mild one. Of the 30 or more cases that passed through my hands this was the only instance of an initial rash. These rashes are said to occur more commonly in some epidemics than others and to occur in two forms, the diffuse scarlatinal and the macular or measly form, the common site being the lower part of abdomen and inner side of thighs.



Case 15 is of special interest. 1. The abortive type may have been due to successful revaccination three days before the initial symptoms or to the epidemic exhausting itself and the cases becoming of a milder type. I am inclined to the latter view. 2. Arthritis is stated to be not uncommon in small-pox, and may go on to suppuration.

Of complications, pharyngitis and dysphagia were present in the severe cases, together with a certain amount of laryngitis and bronchitis. The fact that we escaped serious complications such as corneal ulcers, iritis, otitis media, &c., I attribute in no small measure to the care and attention the nursing orderlies gave to keeping the eyes, nose, and mouths of the patients clean and free from all noxious matter.

Differential diagnosis.—Small-pox in its mild form might easily be confused with chicken-pox and also pustular acne.

From *chicken-pox* the eruption can usually be distinguished by its distribution. In small-pox the eruption tends to be more profuse on the face, back, and upper portions of the chest, and on the extremities. In chicken-pox the eruption is most profuse on the body and is scantier on the face and extremities.

In small-pox the eruption is usually in the same stage of development in the same part of the body and does not come out in successive crops, as in chicken-pox. Further, the papules have a hard shotty feel and do not develop so quickly as in the latter disease. Moreover, in the vesicular stage the characteristic small-pox vesicles are circular in shape, distinctly loculated, and usually umbilicated, are deep-seated, with definite inflammatory areolæ around their bases; whereas the chicken-pox vesicles are often ovoid, with a more rounded apex, are superficial, and are never loculated, and seldom umbilicated, and never have the definite areolæ and infiltrated bases that the small-pox vesicles have.

In *pustular acne* one finds the eruption in all stages of development in the same site. Further, acne pustules tend to come out in successive crops and the presence of scars of previous attacks is generally noticeable.

Prognosis.—The majority of those attacked with confluent variola are said to die. To this statement I would add that early severe erythema of the face, accompanied by œdema, is indicative of a severe confluent type. The subsidence of the œdema of the face on the eighth or ninth day—i.e., at the height of the disease, as occurred in Case 9—I took to be a good sign, especially when followed by a gradual fall in temperature.

When the fever increases after the appearance of the pustules it is a bad sign, but a subnormal temperature after the appearance of the pustules, as occurred in Case 1, is more grave, being indicative of a poor resistance.

Treatment.—There is no specific, and treatment has to be on the general lines of any fever. For the burning and itching caused by the eruption I found frequent bathing with a 2 per cent. carbolic lotion had a very soothing effect. The eyes should be frequently irrigated with warm boric lotion and attention given to the nose and mouth. When crusts begin to form oily applications seem best, such as weak carbolised oil or vaseline.

For the odour of the disease, which is very objectionable both to the nurses and patient, I found a little oil of eucalyptus sprinkled about the floor and bedding had the effect of both drowning the obnoxious smell and giving a fragrant and soothing atmosphere for the patient's respiratory passages, which are frequently more or less affected.

In conclusion, I should like to acknowledge my gratitude to my commanding officer, Lieutenant-Colonel Burke, D.S.O., R.A.M.C., for the valuable aid he rendered me in arriving at a diagnosis in the early cases.

Reviews and Notices of Books.

WAR AND HOUSING PROBLEMS.

I. *Welfare and Housing: A Practical Record of War-time Management.* By J. E. HUTTON, Manager of the Labour and Catering Department of Vickers, Ltd.; Member of the Food Investigation Committee of the Ministry of Munitions. With 12 illustrations from photographs and two plans. London: Longmans, Green, and Co. 1918. Pp. viii. + 192. Price 5*s.*

II. *Village Life After the War.* London: Headley Brothers, Ltd. 1918. Pp. v. + 118. Price 1*s.* net.

III. *New Towns After the War: An Argument for Garden Cities.* By "NEW TOWNSMEN." London: J. M. Dent and Sons, Ltd. 1918. Pp. 84. Price 1*s.* 8*d.* net.

I. War conditions have made welfare work an urgent necessity. The aggregation of vast numbers of people in munition factories and community centres has led to the establishment of welfare management as a department of social science and an indispensable section of the art of human betterment. It is now realised that welfare management must be conducted in accordance with physiological and psychological principles as well as on strict business lines. War, with its concomitants and consequences, has compelled the nation to elaborate special departments of prophylactic and remedial assistance which can collectively be grouped under the general heading of social medicine. Mr. Hutton's informing manual furnishes an interesting and practical work which will prove of real service to many medical advisers and will do much to stimulate endeavours for the establishment of medico-sociological work for factory workers on a scientific and effective foundation. The descriptions are based on actual experiences in connexion with the various war industrial centres conducted by the firm of Vickers, Ltd. The work formulates the essentials of welfare supervision, explains measures for temporary and permanent housing, and describes principles and practices governing catering, the conduct of canteens, and the estimation of food values. There are also suggestive chapters on motor transit, amusements, works police, and industrial unrest. The information in the chapter on Hospitals and Medical Service is of particular medical interest, and we also direct attention to the helpful section which deals with welfare work from the women's point of view. There are appendices dealing with certificates of fitness, sanitary accommodation, washing conveniences, ambulance and first-aid arrangements, the treatment of minor injuries, and provision for the supply of drinking-water. The illustrations and plans add much to the interest and value of the book. The work is one which every welfare supervisor will do well to study, and we commend it to all medical practitioners called to advise in any capacity regarding personal hygienic management and sanitary arrangements for community benefit.

II. Among the problems of after-war reconstruction a prominent place must be allotted to questions dealing with rural developments. The complexity and far-reaching importance of social, economic, and hygienic considerations in the betterment of human conditions in country districts are forcibly brought out in the Reports of Conferences on the Development of Rural Life convened by the Rural Organisation Council. The reports merit the serious study of all medical officers of health and others interested in the protection and progress of human health and happiness in country districts. Amongst the subjects discussed are means for the increased production of food, legislative and other measures for the organisation of satisfactory conditions particularly in view of demobilisation, wages, rural credit, coöperation, housing, rural planning, de-urbanisation of industries, education and social amenities, provision of village community centres, protection of commons, provision of open spaces and the arrest of rural disfigurement, rural continuation school buildings, and the development of country industries and handicrafts. Such a brief enumeration is sufficient to indicate the importance of the volume of transactions and the aims of the Rural Organisation Council, the headquarters of which are at 25, Victoria-street, Westminster, London, S.W. 1.

The objects of the Council are thus defined:—

"To improve the conditions of Rural Life in Great Britain, and for those purposes to federate and secure the harmony and coöperation of any subscribing and approved non-party societies or associations concerned with rural life, while preserving their separate individuality and independence, and in particular to assist the publicity work and the financial needs of the federated societies and associations and generally to take steps for the promotion of agricultural prosperity in Great Britain, and to pursue or carry out any other lawful objects or functions which may conduce to the above objects or any of them."

III. An informing brochure has been issued to expound the aims of the National Garden Cities Committee, the headquarters of which are at 19, Buckingham-street, Strand, London, W.C. 2. This body advocates the building of new towns and the development of existing small towns as a means of dealing with the after-war housing problem and as part of the process of industrial, agricultural, and social reconstruction. The pleas, arguments, ideals, and admonitions set forth are worthy of the consideration of all concerned in the search for a solution of the housing problem in accordance with sound planning, æsthetics, and the maintenance of a high standard of human health, happiness, effectiveness, and community spirit.

Abnormal Forms of Tetanus. (Military Medical Manuals.) By MM. COURTOIS-SUFFIT and R. GIROUX. Edited by Surgeon-General Sir DAVID BRUCE and Captain FREDERICK GOLLA, R.A.M.C. London: University of London Press. 1918. Price 6*s.* net.

THE universal adoption during the present war of prophylactic injections of antitetanic serum as soon as possible after a wound has been received has modified in many cases the symptoms of tetanus. Man has been converted from a highly susceptible to a resistant subject, and as a result local tetanus, formerly a very rare type, now constitutes nearly 20 per cent. of the total number of our cases of tetanus. It is to the credit of the authors that they were able to give the first systematic account of the various forms of abnormal tetanus. It is curious, however, that their experience does not furnish many examples of the purely local form, never spreading beyond the group of muscles originally affected, with which we are so familiar in this country. The majority of the types described by them would be classified as cases of generalised tetanus with a local onset. In view of our later experience, much of the space devoted to a rather discursive and not very authoritative account of the general therapeutics of tetanus might have been with advantage given to a discussion of the pathology and the differential diagnosis of local tetanus from the various types of contracture. The book is of considerable value as a clinical exposition of some little known types of tetanus. The editors, Sir David Bruce and Captain Golla, sound a not unnecessary note of warning on the subject of treatment, recent statistics giving little encouragement to the uncritical optimism that is prevalent in many quarters.

Le Paludisme Macédonien: Caractères Cliniques et Hématologiques, Principes de Thérapeutique. Par P. ARMAND-DELILLE, G. PAISSEAU, P. ABRAMI, et HENRI LEMAIRE. Paris: Masson et Cie, Editeurs Libraires de l'Académie de Médecine. 1917. Price 3 frs. 50. *Malaria in Macedonia.* Translated by J. D. ROLLESTON, M.D. Edited with a Preface by Sir RONALD ROSS. London: University of London Press, Limited. 1918. Pp. 115. Price 6*s.*

THE work on malaria published in this country during the last year has established for most of us the following three things: (1) that only by a microscopic examination can a diagnosis of malaria be made; (2) that it is unscientific to speak of the treatment of malaria, when we should distinguish between the treatment of simple tertian and malignant tertian malaria; and (3) that it is essential when we speak of "curing" malaria to state clearly what we mean by the word "cure." "Cure" as regards malaria strictly means that the parasites in the body have all been killed. As we have at present no means of ascertaining whether this is so or not, our only means is to examine the blood periodically and determine whether or no parasites are present in the blood. A rigor by itself is no evidence of malaria, not even in a case that has shown parasites, for a reference to the records of the work done in this country will show, even in malaria cases, rigors without parasites in

the blood. The nature of these remains to be determined just as much as the nature of any other rigor of unknown origin. If we are to judge the value of the curative treatments for simple tertian malaria recorded in the last chapter of this book we must have these facts in our mind. We see no means of deciding whether any of them are of use, when nothing is said about the observation period. We cannot observe patients for ever, but we can only estimate the value of "Job's treatment," "the Italian treatment," "Carducci's treatment," or "Ravaut's treatment" (p. 109) if we know for how long these treatments have "cured" their cases, whether for one day, one week, one month, or one year. As regards the treatments themselves the reasons for them are not given, and we are asked to accept an *ipse dixit* as equivalent to a conclusion based on facts. In the important matter of curative treatment, as distinct from the treatment of a paroxysm, we find no guidance in this work.

The authors regard primary malaria as invariably due to the malignant tertian parasite and secondary malaria as due to the simple tertian malaria parasite. Not all observers in this country will agree with their view, for infections originating in this country where the first attack was due to the simple tertian parasite have been recorded. It will be gathered from these remarks that we do not consider all the authors' statements proved, but as a basis for further study the book will be found of great use, and it should be in the hands of all who are called upon to treat malaria. The work is marked by an admirable clearness of style, and the summary of clinical symptoms due to the different species of parasite is excellent.

I. *Minor Maladies and their Treatment.* By LEONARD WILLIAMS, M.D.Glasg., Physician to the French Hospital. Fourth edition. London: Baillière, Tindall, and Cox. 1918. Pp. xi. + 402. Price 7*s.* 6*d.* net.

II. *The Art of Keeping Well.* By RONALD CAMPBELL MACFIE, M.A., M.B., C.M., Hon. LL.D. Aberd. With one colour and three half-tone plates. London: Cassell and Company, Ltd. 1918. Pp. xi. + 244. Price 6*s.* 8*d.* net.

I. Dr. Leonard Williams's book, first published in 1906, is now in its fourth edition. The author exposes in a preface the inadequacies of existing methods of medical training which permit the young practitioner to enter on his professional life incompletely equipped for dealing with derangements which, in all probability, will form the major part of his practice, and thus fully justifies what follows in his book. The work deals with colds, coughs, and sore-throats, indigestion, constipation, diarrhœa, vomiting and giddiness, rheumatism, neuralgia, and headache, goutiness, and minor glandular insufficiencies. There are informing chapters on the maintenance of general health, the care of old age, the supervision of mentally deranged subjects, and the use of certain drugs. Dr. Williams possesses a vigorous style and writes with directness and humour.

II. Dr. Macfie has written an interesting and serviceable book on the principles of health, designing his work for non-medical readers. But it will prove of service to medical advisers who are called upon to explain physiological facts to their patients, or to lecture to nurses, ambulance and first-aid classes, as well as to those called to direct the instruction of welfare workers. The subjects dealt with relate to man's food requirements, the dietetic characters of the principal foodstuffs, digestion and absorption, the mechanism of breathing, air and climate, muscular activity, sleep, the care of the skin, breathing and clothing, and also the hygiene of the home. There are helpful chapters on alcohol, tobacco and drugs, common minor ailments, and the relationship of body and mind. Dr. Macfie has the knack of expressing in attractive form guiding truths and governing principles.

JOURNALS.

The British Journal of Children's Diseases. Vol. XV., April-June, 1918. Edited by J. D. ROLLESTON, M.D.Oxon.—Mr. R. Thompson writes on cases of urinary disease in children, which, he states, are not so common as in adults owing to the absence of obstructive causes such as enlarged prostate or pregnancy. Obstructive lesions, however, may occur as a sequel to tuberculous peritonitis or abdominal adenitis, which may cause kinking or compression of the ureter. Mr. Thompson records cases of pyonephrosis due to aberrant blood-vessels, renal calculi, or fibrous adhesions kinking the ureters in children aged from 8 to 13 years, and draws attention to the fact that in children symptoms of renal calculus may occur on the side which is not affected. The commoner symptoms of vesical calculus in children, in his experience, are frequency of micturition and incontinence. The prognosis is not good, and lithotrity should always be preceded by cystoscopy. The prognosis of traumatic stricture in children is also unfavourable; if possible the stricture should be excised and the ends sutured.—Dr. E. Bronson contributes a paper on Erythema Nodosum Associated with Tuberculosis, and records a case of acute pleural effusion in a boy, aged 9 years, suffering from erythema nodosum. Dr. Bronson concludes that the supposed relationship of erythema nodosum to acute rheumatism lacks clinical support; that the sequence of fairly well-defined prodromal stage, often with sore-throat, eruptive stage, and slow convalescent period, supports the theory of a specific disease of mildly infectious type; that the possibility of it being a non-specific cutaneous manifestation of general toxæmia must be considered; that the strong reaction to tuberculin and the clinical association of the two conditions are in favour of the tuberculous etiology. Finally, that the etiology of erythema nodosum has not yet been established.—In his article on the nature of von Jaksch's disease Dr. Gordon Ward adopts the following hypothesis. The disease is a reaction of the infantile blood-forming organs to infection. The actual infecting organism is not known, and is not necessarily specific. The particular reaction is only produced when rickets is present. There are therefore two essential factors—viz., infection as an exciting factor and rickets as a predisposing factor.—Dr. H. W. L. Barlow, in his notes on small-pox in childhood, gives an account of the recent outbreak of small-pox in the East-End of London. It was largely an affection of childhood. Of the first 30 patients 14 were under 15 years of age. Of these, 5 had never been vaccinated at all or only after the commencement of the actual illness, and 9 were vaccinated for the first time in the incubation period. The combination of a considerable degree of infectivity with a rather low virulence seems to have been a characteristic of the epidemic. Only one case, a girl aged 6½ years, died.—"Can the Clinical Manifestations of Congenital Heart Disease Disappear with the General Growth and Development of the Patient?" is the title of a note by Dr. Parkes Weber, who quotes two recent cases from the *Guy's Hospital Gazette* to show that such an eventuality is possible, and relates a similar case in a boy who used always to be cyanotic and had a tremendous thrill and a murmur audible at a slight distance from the chest. His development, however, was good, and he was ultimately passed for military service, nothing abnormal being found by the examining doctor. Dr. Parkes Weber also contributes a note on erythema iris in a boy, aged 6 years, in whom the eruption consisted of three patches, the largest in front of the trunk, and a smaller one on each side.—Mr. P. B. Roth relates a case of hæmophilia with effusion into the knee-joints in a boy, aged 6½ years. The condition at first was regarded as tuberculous, but the correct diagnosis was established by a history of hæmophilia and by the following X ray findings which Mr. Roth considers pathognomonic of hæmophilia. There were extensive wearing away and absorption of the cartilage and bone of the femur, tibia, and patella, as well as the formation of new bone at the edges and a deep shadow surrounding the bones, obviously due to distension of the synovial cavity with blood. To obviate ankylosis Mr. Roth proposed to apply two "knee-cages" to limit the movements of the joints and so prevent further attacks of effusion, and also to give regular doses of thymus gland and calcium and magnesium salts.—Dr. James Burnet records a case of pregnancy in a girl, aged 14, in whom menstruation had commenced at 12. The father was a boy of about the same age. Pregnancy lasted 268 days and resulted in the birth of a healthy male infant weighing 9 lb.—Drs. Marcel Labbé, Targhetta, and Ameuille publish the first two cases of infantile kala-azar in France. The patients were Serbian refugees, aged 6 and 9 years respectively, living at Nice, and had probably been infected by a pet dog.—Dr. E. Jeanselme and Mme. Chatelain describe an epidemic of familial syphilis in which a soldier home on leave infected his wife and indirectly five other members of the family.—In a note on a fifth cusp and hereditary syphilis Dr. C. Mantoux contests Sabouraud's claim that a fifth cusp on the inner surface of the first upper molar is invariably associated with inherited syphilis. In 7 cases with this fifth cusp examined by Mantoux none presented any history or signs of inherited syphilis, and the only two who had a positive Wassermann reaction were the subjects of acquired syphilis.—The abstracts from current literature deal with diseases of the blood, circulatory system, urogenital system, dermatology and syphilis, and school hygiene.

A RECENT Order of the Scottish Local Government Board makes the notification of ophthalmia neonatorum compulsory as from Nov. 1st, 1918.

Reports and Analytical Records
FROM
THE LANCET LABORATORY.

DRIED MILK (AMBROSIA BRAND).

(AMBROSIA LIMITED, BRIGHTON CHAMBERS, DENMAN-STREET, LONDON BRIDGE, S.E.)

THE importance of dried milk has recently been emphasised in a report of the Local Government Board reviewed in our columns.[1] The consensus of opinion of the authorities quoted in that report is in favour of the use of dried milk in infant-feeding, when, of course, breast-feeding is not available, and it appears that the physical and chemical changes produced by the processes used in the preparation of dried milk so alter its character that it is better assimilated by the infant than is ordinary cow's milk, whether raw, boiled, or sterilised. Further, even if the milk powder is not entirely soluble on reconstitution, its digestibility is not affected except for good. This probably is largely due to the fact that the casein of dried milk separates on the addition of gastric juice as a fine, soft curd, and not in lumps, as is the case with ordinary cow's milk. Such conclusions are of the utmost importance, since they justify the drying of milk and the conservation in this way of our available milk-supplies, well known to be unsatisfactory, because milk in the fluid state is perishable and liable to contamination on its way from its source to the consumer. The specimen submitted to us by the above company presents all the characteristics of a dry milk powder prepared from clean milk by an effective drying process and under strictly hygienic conditions. Our analysis gave the following results: Moisture, 1·60 per cent.; proteins, 28·71 per cent.; fat, 29·29 per cent.; milk sugar, 35·20 per cent.; mineral matter, 5·20 per cent. It will be seen that the proportion of fat is most satisfactory, and in general the results show that in this powder is preserved the complete food value of cow's milk of good quality. We understand that the dairy and works of this company are situated in Devonshire, and that, although primarily the manufacture was installed in order to supply the troops with milk food, a balance is reserved by permission of the War Office for the purpose of infant feeding.

"CLAROCIT."

(ARNOLD AND SONS, GILTSPUR-STREET, LONDON, E.C.)

"Clarocit" consists of a round tablet of a soap-like nature, and has been designed for preventing the condensation of moisture on laryngoscopic mirrors and lenses. The procedure is simple. Some "clarocit" is taken up by the moistened finger or suitable cloth and rubbed on the glass surface. A dry film is obtained when left for a few moments, which is removed by polishing. The method, according to the trials we made, answers admirably, condensation of moisture from, for example, the breath being completely prevented. The price of the tablet seems to us somewhat excessive, but of course the difficulties of all production are to-day abnormal. There are two sizes, 2*s.* 6*d.* and 5*s.* respectively, and the tablet we examined weighed not more than ¼ oz. A very small quantity, however, suffices for the purpose.

HAY FEVER OUTFIT.

(DUNCAN, FLOCKHART, AND CO., EDINBURGH. LONDON OFFICE, 155-157, FARRINGDON-ROAD, E.C.)

This outfit comprises (1) the ophthalmic test, and (2) the vaccine prepared from the pollen of Timothy grass. The diagnosis is indicated by the former and treatment by the latter. A drop of the toxin solution is instilled on the eyeball of the patient, and the strength of the solution (supplied in capillary tubes) which first causes the ophthalmic reaction is noted. The dose of the pollen vaccine is based on this indication, and is one-third to one-half of the number of units of ophthalmic solution which first produces a reaction. Thus a patient giving a diagnostic reaction with 25 units would receive 10 to 15 units of the vaccine hypodermically. The vaccine is contained in a graduated series of doses in ampoules, providing a good range for the varying indications of the patient.

[1] THE LANCET, April 6th, 1918, p. 509.

Both ophthalmic reaction solution and the pollen vaccine are prepared in the laboratory of the Royal College of Physicians, Edinburgh, in which has also been prepared for some time a tuberculin described by Professor Beraneck, director of veterinary medicine at Neuchâtel. This tuberculin is a mixture of equal parts of a filtered culture of tubercle bacilli on an albumose-free medium, and a phosphoric acid extract of bacillary bodies. It is claimed that this tuberculin is less toxic than, and equally efficacious to, Koch's tuberculin.

FLY-QUIT PRODUCTS.

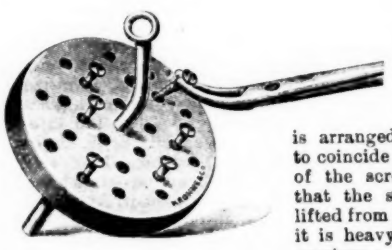
(LAWSON AND COMPANY, LIMITED, ST. PHILIP'S, BRISTOL.)

We referred to these preparations in this column in THE LANCET of Sept. 1st, 1917, since when further attention has been given to increasing their efficiency and convenience as muscicides. They are non-poisonous, and "fly-quit" spray has been found useful in rendering meats and uncooked food-stuffs fly-proof without affecting the normal flavour of the food material in any objectionable way. The concentrated preparation forms an emulsion with water which is non-staining and of broad "wetting" power. As a muscicide the toxic action is slow, so that the flies do not as a rule drop dead amongst the food or, indeed, indoors if they are free to escape. Another preparation is "fly-quit" oil, which is used against blow flies out-doors; and, lastly, there is "fly-quit" emulsion, which has been successfully employed for killing both flies and larvæ in manure and refuse heaps. The preparations are free from offensive smell and have the pleasant characteristics of wood-tar.

New Inventions.

A "SCREW STAND" FOR USE IN BONE-PLATING.

IN plating bones according to the principles of Lane I have found much time wasted and much annoyance caused by difficulty in seizing hold of each screw with the screw-holder preparatory to inserting it in the bone. I have therefore devised a screw stand which does away with all the difficulty (see figure). It is arranged at an angle to coincide with the neck of the screw-holder, so that the screws can be lifted from it in a second; it is heavy and will not overturn; it is lifted straight out of the steriliser by the ring and placed on the instrument table, so that all chance of contamination of the screws is avoided. Moreover, in seizing the screws only one hand is required, and in a typical operation several minutes are saved. The stand is made by Messrs. Krohne and Sesemann.



PAUL BERNARD ROTH, F.R.C.S.,
Senior Surgeon, Kensington General Hospital;
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THE LANCET, VOL. I., 1918: THE INDEX.

OWING to the continued shortage in the paper-supply, the Index and Title-page to the volume of THE LANCET which was completed with the issue of June 29th have been printed separately, and copies have been supplied gratis to those subscribers who have, up to August 14th, intimated to us their wish to receive them. Other subscribers will be similarly supplied, so long as the stock remains unexhausted, on application to the Manager, THE LANCET Office, 423, Strand, London, W.C. Such applications should be sent in at once.

THE LANCET.

LONDON: SATURDAY, AUGUST 17, 1918.

The Small-pox Danger.

IN a report to the Local Government Board on the incidence of small-pox throughout the world in recent years Dr. BRUCE LOW has given a detailed account of the behaviour of this disease which shows its presence in almost every quarter of the globe. He sounds a warning note as to the dangers likely to arise in this country from the continued neglect of vaccination and revaccination by a large section of the public. The accumulation at home of this unprotected material is attended with considerable risks at the present time, and these are likely to become still more serious at the end of the war when large bodies of troops, as well as other persons, will arrive in this country from areas in which small-pox is endemic, or where recent epidemics have been occurring. This is no alarmist pronouncement but a justifiable and probable surmise.

In the present day many of the younger generation of British medical practitioners have had little or no opportunities of becoming acquainted with small-pox. Under normal conditions the occurrence of an imported case in a well-administered district is swiftly and successfully dealt with by immediate isolation of the patient and segregation of the contacts, along with vaccination and revaccination. But it is not so easy to deal with cases in some other countries. There are certain races among whom small-pox is almost always present, and this is the case with the Arabs inhabiting the region from Morocco to the western shores of the Persian Gulf, among whom small-pox as well as exanthematic typhus is always occurring, and who, when coming from the interior to coastal towns or to depôts on the caravan routes, convey small-pox to the susceptible individuals who may come in contact with them. An instance of this is given in a communication which we publish in our columns by Lieutenant G. H. MEAD, R.A.M.C.(T.C.), entitled "Notes on a Recent Epidemic of Small-pox in the Field." Here is given illustration of the danger from the visits to camps of friendly Arabs selling their fruit and vegetables to our soldiers and camp followers. Lieutenant MEAD describes a localised outbreak, comprising about 30 cases, of which he had the supervision, and he gives some interesting clinical facts concerning the patients under his charge. We infer that vaccination and revaccination of men in our expeditionary forces have not been so persistently carried out as in former days. Incidentally, Lieutenant MEAD shows the danger of small-pox to those who have reached middle age, and who, though vaccinated in infancy, have not been revaccinated, and have consequently long lost their previous immunity. He also illustrates the folly of postponing revaccination till after exposure to the infection and therefore too late to arrest the development of the disease; and, further, he shows the serious nature of a small-pox attack in an unvaccinated person who has been exposed to a virulent strain of the infection. Lieutenant MEAD'S "Notes" are worth study by those who wish to keep themselves posted up in the possible occurrence of small-pox, first in our expeditionary and secondly in our home communities, wherever somewhat analogous conditions occur.

Dr. BRUCE LOW, in his report, refers to the disastrous pandemic of small-pox which began during the Franco-German war, and which spread over the whole of Europe, extending to the western hemisphere, and causing, in Europe alone, about half a million of deaths in three or four years, of which number not far short of 50,000 occurred in England and Wales. He is evidently alive to the possibility of such sinister history repeating itself. The existing conditions of a great European war, with a battle-front from Flanders to Mesopotamia, involve countries in which small-pox is already present; in addition to this we have to reckon with the displacement from their homes of large numbers of the poorer classes, as well as refugees, deported populations, and prisoners of war, many of whom are unvaccinated; and their aggregation takes place under unwholesome conditions of over-crowding, distress of mind, and privation, with consequent lowering of vitality. Now all these things are likely to intensify the type of the disease. Should such virulent strain of infection be introduced into this country, with its accumulations of unvaccinated children and unrevaccinated adults, it is more than probable that a serious epidemic would follow. The situation would be further aggravated at the present time if the epidemic began in the absence on military service of many of our medical officers of health, public vaccinators, inspectors of nuisances, vaccination officers, and other officials, who in ordinary circumstances could be trusted to deal successfully with the early manifestations of a small-pox epidemic. The public who have in a large measure lost their former dread of small-pox in consequence of its comparative disappearance from our land, and have, therefore, neglected vaccination, would almost certainly suffer severely. Dr. BRUCE LOW'S report comes as a timely reminder that with all our boasted progress in sanitation and preventive medicine we cannot afford to ignore the danger of small-pox; nor can we continue as a nation to neglect the only prophylactic against it known to medical science—namely, vaccination and revaccination.

40 D.

THE Regulation under the Defence of the Realm Act commonly known as 40 D having now been in force for some months, it is not too early to begin to inquire into its value as a measure of public health, for evidence is becoming available on which to base a judgment. It must be as a public health measure that the Regulation stands or falls, since it was introduced as such, and was not held out by its promoters either as a device for making promiscuous sexual intercourse safe for men or as a form of duress for women of loose morals.

The public health intent of the Regulation is made evident from its phrasing, which runs as follows:—

"No woman who is suffering from venereal disease in a communicable form shall have sexual intercourse with any member of H.M. Forces or solicit or invite any member of H.M. Forces to have sexual intercourse with her."

Now, in the first eight charges under the Regulation in various parts of the country, from Manchester in the north to Portsmouth in the south, four convictions were obtained and penalties of two to six months' imprisonment with or without hard labour inflicted. In four cases the accused was acquitted, although in one of these the woman was committed to prison for a month for keeping a brothel. Last week, at a London police court, the solicitor acting for the Commissioner of Police said that so far as his experience went prosecutions under the Regulation had not turned out satisfactorily; of five cases conducted by himself all had failed. The difficulty, he added, was due to the time elapsing between inception of the disease and examination of the woman. In the case actually before the court no hardship, he said, could be alleged, as the woman was a prostitute. In a similar case brought early last month before a London police court a charwoman was fined for assisting in the management of a disorderly house, although the magistrate acquitted her under Regulation 40 D after the gaol medical officer had reported that he could find no evidence of disease. We observe, therefore, that up to the present time, in a large proportion of cases, it has proved impossible to establish the existence of "venereal disease in a communicable form," although in some at least the women were demonstrably living a disorderly life. It may without unfairness be concluded that a searching bacteriological examination, involving the use of the vaginal speculum and the expression of secretion from the urethra and cervix uteri, may fail to establish evidence of infection in cases where clinically it was almost certainly present. And this conclusion is entirely in harmony with the admitted difficulty—some experienced clinicians go so far as to call it impossibility—of curing gonorrhœa in the female. Quite apart from the fact that the woman may be acting merely as carrier of infection without at the time clinically suffering herself from venereal disease, it may frequently happen that an infective spot is present in the genital tract which is beyond the reach of the most exhaustive examination. Put shortly, the coitus test is more delicate than the bacteriological. With regard to the latter test, Regulation 40 D runs:—

"A woman charged with an offence under this regulation shall ..... be remanded ..... for the purpose of such medical examination as may be requisite for ascertaining whether she is suffering from such a disease."

But in practice no medical examination suffices to ascertain the presence of infection, and this negative aspect in itself, we believe, discredits the Regulation as a public health measure. Also difficulty may arise in regard to positive medical evidence. In a case where the woman is shown to be infectious, and where sexual intercourse has taken place, the length of time elapsing between inception of the disease and examination of the woman, adduced by counsel as a legal difficulty, may leave the medical examiner himself in doubt as to which party infected the other. Either the man alone, or both the man and the woman, may have been suffering from communicable disease at the time they met, and the statement of either party as to the date of possible infection is evidently always to be taken with reserve. Great probability may exist as to the precise nature and order of the events, and yet that degree of certainty may not be present which will enable the medical examiner to speak without reserve in a matter which involves such serious issues.

We have confined our criticism thus far to the purely public health aspect of Regulation 40 D. We agree entirely with Mr. MACPHERSON'S recent statement in the House of Commons that the War Office ought to do everything to preserve the health of the troops, but we are not sorry to find wanting as a public health measure a regulation which has aroused such widespread opposition from all who wish to see the law dispensing even-handed justice. Solicitation is difficult enough to define, and should be abolished as a legal offence, unless the attitude of intending to be solicited is recognised as a contributing offence. With regard to the actual duties of the examining doctor, examination of a woman, in her own interest, for the relief of present suffering or future disability, and in order to carry out the treatment found to be necessary—however disagreeable or painful the episode may be to the patient—is not in itself an idea foreign to medicine. But the position is essentially altered when examination is not primarily or necessarily made for the purpose of consequent treatment. We believe that the cause of public health, which the medical profession has increasingly at heart, will not be advanced by any legal measure which does not commend itself to the moral sense of the majority; but whichever way that sense may work in respect of Regulation 40 D, the enactment appears to be condemned by its own futility.

The Medical Side of Chemical Warfare.

A SERIES of reports which demand more than passing comment have just come into our hands. They are issued by the Chemical Warfare Medical Committee, a body appointed last January by the Director-General of the Army Medical Service to advise him on the scientific medical aspects of chemical warfare. The committee as now constituted has Professor LEONARD HILL, F.R.S., as chairman, Dr. J. S. EDKINS as secretary, the other members being Mr. J. BARCROFT, F.R.S., Professor W. M. BAYLISS, F.R.S., Professor A. E. BOYCOTT, F.R.S., Dr. H. H. DALE, F.R.S., Sir WALTER FLETCHER, F.R.S., Dr. J. S. HALDANE, F.R.S., Lieutenant-Colonel J. C. MEAKINS, C.A.M.C., Major H. S. RAPER, R.A.M.C., and Dr. F. SHUFFLEBOTHAM, with corresponding members in France and Italy. The series consists of eight reports issued by this committee during the present year dealing with various phases of the pathology and treatment of gas poisoning—matters which have hitherto been kept secret. A number of the reports are still to

be regarded as confidential, while the remainder may be treated in any way likely to advance the interests of the medical public.

It has long been evident to us that records of this sort were necessary if the medical profession was to be of full service to the country, and the knowledge that official information on the pathology and treatment of gas poisoning is forthcoming, and that there is a definite organisation for dealing with it, will do much to allay anxiety. The subject of gas poisoning, considered as a part of medical knowledge, presents novel features in scientific development. For some decades pathological inquiry in this and other countries had been directed, and with good reason, mainly towards bacteriology and its kindred subjects, such as protozoology. These, together with the always essential studies of morbid anatomy and morbid histology, occupied the minds of our principal pathological workers when, in April, 1915, gas warfare commenced. Casualties of an unconsidered type threatened whole armies and the fate of nations, and to the medical profession the world looked for counsel. At first, gas warfare consisted simply in the use of pulmonary irritants, the treatment of its effects being dependent upon the answers to a few simple questions, such as the following: "How much oxygen does this man need to preserve life; how can this be reduced to a minimum; how much do the pulmonary conditions admit of his obtaining, and for how long?" The whole pathology and treatment of gas poisoning had clearly to be worked out from first principles. In this country happily we are rich at the moment in authorities on the physiology of respiration, and of these experts the Chemical Warfare Medical Committee appears to have made full use.

The novelty of the subject has had a striking effect upon the composition of the Committee itself. It has brought into prominence a school of young clinicians, all of whom had, we believe, published scientific work before the war. Such men as Colonel MEAKINS, Captain G. H. HUNT, and Captain R. A. PETERS had been known to us as producing sound work in our medical centres; it is valuable to have such observers associated with our specialist workers. For it is essential that such a novel subject as the treatment of gas poisoning, and one which is ramifying in so many directions and which has relations with our own production of materials of war, should be in the hands of a young and capable school. Continuing activity is evident from the fact that some of the pamphlets which we have received are already obsolete in parts and undergoing revision. We trust that it may be possible to issue No. 1, dealing with the treatment of gassed cases at the front, to medical officers as soon as it has undergone the necessary revision. For practitioners in England a knowledge of the early pathology and treatment of these cases is eminently desirable, and we are confident, therefore, that as soon as secrecy can be relaxed the committee will make use of their freedom. In the meantime we advise all practitioners who are interested in gassed cases to apply to Dr. EDKINS at the Department of Physiology, Bedford College, London, N.W., for such numbers of the series as may be had.

Annotations.

"Ne quid nimis."

THE BRITISH SCIENTIFIC PRODUCTS EXHIBITION.

IT is early yet to deal in detail with the splendid collection of British scientific products now on view at King's College, since at the informal opening on Monday last the work of arranging the exhibits had not been completed. There was enough, however, already on view to show that the chief purpose of the exhibition promises to be fulfilled in a most gratifying way. The belief is amply justified that such an exhibition at the present time will have a most stimulating influence upon scientific and industrial research and upon manufactures, and that the highest permanent interests of the nation will thus be promoted. The products shown come under three categories, as follows:—(1) Products imported chiefly from enemy countries before the war, but now made in this country; (2) products in the manufacture of which we excelled in this country even before the war and exported to enemy countries; and (3) products in the manufacture of which considerable developments have taken place during the war. A very large number of the exhibits have a direct medical interest, as, for example, the surgical, bacteriological, and pathological appliances, including X ray apparatus in Section 8, the optical apparatus in Section 4, and the pharmaceutical and medicinal preparations, including the organo-metallic preparations shown in Section 1, comprising chemical products and processes. The exhibition was formally opened, after an impressive speech, by Lord Sydenham, President of the British Science Guild, on Wednesday last, when the stalls presented a remarkable object-lesson as to what British scientific workers are capable of doing. With the object of ensuring an intelligent interest in the several exhibits, men well grounded in their subject attend the stalls to give any explanation which is sought, and from time to time short lectures and addresses will be given dealing with the departments and features of the exhibition. By this excellent plan public attention should be effectively directed to the vital necessity of developing our scientific industries and of applying to them the results of scientific research. Altogether the idea is admirably conceived, and the occasion will doubtless lead to results of far-reaching importance to British scientific manufactures. The exhibition will be kept open until Sept. 7th, and thus presents a remarkable opportunity to the public of obtaining ocular demonstration of British scientific capability.

MALARIA IN FRENCH WEST AFRICA.

DAKAR is a very malarious port, numerous infected mosquitoes invading the ships as they enter the inner harbour. Each of three French ships which lay there for four months had about 400 cases on board, and a Senegal regiment had an enormous number, 97 per cent. of its strength, attacked. Another ship which passed there the latter half of two consecutive years had in the first period 169, in the second 473 cases. The worst month in each year was October. Of the first attacks, 35 per cent. were tertian, 33 per cent. remittent, while 24 per cent. had but a single pyrexia, and these last by no means those with the fewest parasites in their blood. The story is told in the *Archives de Médecine Navale* for July by Dr. Rouche of the French Navy, who, being well

acquainted with his shipmates, was able to recognise those of them who, during the fortnight of incubation, while their temperature was still normal, were about to be attacked, for their faces, he says, were drawn, their complexions leaden, and a yellow colour spread over their eyelids and round their eyes, their conjunctivæ, too, being slightly jaundiced. The importance of the disease is obvious, for on one day in October, at sea, they had 112 men in bed with malarial fever, and the work of the ship could hardly go on. Dr. Rouche notes how dangerous it was to lie at Dakar; a single day's visit in October started a malarial epidemic in the ship. For prophylaxis it is proposed not to keep any ship there, but to send them cruising to and from the Canary Islands. Quinine prophylaxis (4 gr. quinine daily) is recommended, but there are many men who will not take it. The treatment laid down is: on the first day, nothing but a purge, next day 10 gr. quinine morning and evening as long as the pyrexia lasts, and daily till the temperature is well down, with ¾ gr. of arrhenol daily for a week. 10 gr. of quinine are given daily during the apyrexia, and 15 gr. the evening before a relapse is due.

IRELAND AND THE MINISTRY OF HEALTH.

THE announcement made recently by Mr. Bonar Law that the Government did not contemplate including Ireland in the proposed Ministry of Health Bill has taken the Irish medical profession by surprise. Ireland is greatly in need of a development of her public medical services, which could best be undertaken by a Ministry of Health. The habit of omitting Ireland from legislation dealing with matters of health is hard either to explain or justify, but it is a fact that Ireland has not shared in the health reforms of the past 20 years. The National Health Insurance Act does not carry medical benefits in Ireland. There is no provision for the medical inspection of schools and school children in Ireland. It is only within the past few months that a Midwives Act has been enacted for Ireland, 16 years later than legislation for England. But it is in the ordinary administration of public health that Ireland lags farthest behind Great Britain. In the whole country there are only two whole-time medical officers of health. In fact, in all matters of health, Ireland is almost an untilled field, or a field only tilled in a few patches. The profession had hoped that with the establishment of a Ministry of Health this backward condition might have received due consideration, and proper schemes of development might have been inaugurated. No doubt there would be differences of opinion as to the best method by which the Ministry of Health should be applied to Ireland, but on the general principle there could hardly have been any difference. The Irish Medical Committee, which represents professional opinion in Ireland, declared itself in favour of the establishment of a Ministry of Health, and the Royal College of Physicians of Ireland has expressed itself in the same sense.

THE PREVENTION OF SIMPLE GOITRE BY ADMINISTRATION OF IODINE.

IN the *Archives of Internal Medicine* for July Dr. O. P. Kimball and Dr. D. Marine have brought forward cogent evidence that "simple goitre is probably the easiest of all known diseases to prevent." Their method is the administration of small doses of iodine. Iodine is, of course, an old and most important remedy in the treatment of goitre, but its prophylactic use seems to be entirely new. A census of the condition of the thyroid gland of all girls from the fifth to the twelfth grades in the schools of Akron, Ohio (evidently a very goitrous district), was made. In April, 1917, 3872 girls were examined, and the condition of the thyroid gland was found to be in percentages as follows: normal, 43·5; slightly enlarged, 49·8; moderately enlarged, 6·3; markedly enlarged, 0·18; adenomas, 1. In May, 2 g. of sodium iodide were given in 0·2 g. doses each school day to a number of pupils in grades from the fifth to the eighth, and 4 g. in 0·4 g. doses to pupils in the higher grades. In November the pupils taking the treatment were re-examined, with the following results. Of 283 normal thyroids on the first examination all had remained normal; 287 small goitres had remained unaltered; 141 small goitres had disappeared; 2 small goitres had increased; 34 large goitres had remained unaltered; and 17 had decreased. On the other hand, in pupils not taking the treatment 637 normal thyroids had remained normal and 259 showed slight goitre; 759 small goitres had remained unaltered, 10 had disappeared, and 103 had increased; 106 large goitres had remained unaltered and 5 had decreased. Thus, not a single pupil with a normal thyroid who took iodine showed an increase, while 26 per cent. of those who did not showed definitely enlarged thyroids—some moderately large goitres. More than prophylactic results were obtained. One-third of the small goitres had disappeared and one-third of the "moderate" showed a decrease of 2 cm. or more. Of 1000 girls who took the treatment only 5 developed any noticeable rash, and this was evanescent. The possibility of producing Graves's disease by the treatment was considered, but no evidence of this was observed. As the results were so definite, when the treatment was repeated in November, 1917, only 2 g. of the iodide were given to the pupils in the higher grades. At the time of making this report it was intended to repeat the treatment in April, 1918.

THE SMALL HEART IN TUBERCULOUS SUBJECTS.

FREQUENT observations on patients sent for radiological examination for the diagnosis of tuberculosis have enabled Dr. Carlo Guarini[1] to collect a large number of cases of the small median heart which is said to be characteristic of tuberculous or pre-tuberculous patients. Rokitanski was one of the first to draw attention to the small size of the heart, which he considered congenital and a sign of infantilism, and Albert-Weil more recently described it as a frequent occurrence in individuals who were tuberculous or predisposed to tuberculosis. Barjon also noticed that the tuberculous were habitually the subjects of a median heart which overlapped only to a small extent, to right and left, the central shadow. According to this observer, the median heart is indicative, if not of tuberculosis, at least of a predisposition to it, while, on the contrary, the existence of a large heart in an individual suspected of pulmonary tubercle is of favourable interpretation. Vaquez and Bordet drew attention to the small narrow heart suspended from a long aorta, the so-called "dropping heart" (*cuore a goccia*), which, however, they regarded as a physiological variety and having no pathological significance, arguing that if it be true that a small vertical heart is often found in tuberculous patients it is

[1] La Riforma Medica, June 22nd, 1918.

due rather to the peculiar formation of their thorax than to the pulmonary tuberculosis itself. Dr. Guarini's observations throw some light on these different views and tend to show that the small heart and the dropping heart are pathological entities in close dependence on the hypo-evolution of the other organs of the individual and on the hypoplasia of its anatomical parts. Of 1300 tuberculous patients subjected to radiological examination he found the small median heart in 68 per cent. of the cases, whereas the dropping heart was only found in 0·13 per cent. He considers that the former is a hypoplasic heart, while the latter, also hypoplasic, is due to an arrest of development of the heart and aorta during extra-uterine life; that both these varieties must be considered pathological entities, having no relation with the atrophic heart found in other morbid conditions, and that their presence is a far more reliable aid than clinical symptoms in the diagnosis and prognosis of tuberculosis. Few practical physicians are likely to take this view. Dr. Guarini has also made observations on the inclination of the heart's axis in the same class of patients. The older anatomists and clinicians fixed this inclination between 55° and 60°; but Moreau, from radiological investigations on 100 normal adolescents and adults, found that in 74 per cent. the inclination varied between 65° and 78°. Dr. Guarini found in 100 patients examined for diseases of the chest the inclination was in 6 per cent. between 20° and 30°, in 37 per cent. between 30° and 40°, in 41 per cent. between 40° and 50°, and in 16 per cent. between 50° and 60°. Thus the angle of inclination varied with the individual, or rather with the morphological constitution of the individual, between 20° and 60°, which may be considered the extreme limits in tuberculous or pre-tuberculous subjects. It should be noted, however, that there may be small hearts whose axis of inclination exceeds the above limits, and these must also be considered pathological, since they represent hearts with incompetent valves or stenotic orifices.

CANADIAN COLLECTION OF MEDICAL WAR SPECIMENS.

FROM 1914 onwards the Canadian Army Medical Service has coöperated with the Army Medical Service in the endeavour to obtain adequate medical records of the world-war. Being, perhaps, familiar with the great collections which the United States possess from their Civil War in the Army Medical Museum at Washington, the C.A.M.C. has steadily been working ahead to obtain material of all sorts which will worthily illustrate the medical aspect of the war. In 1915 the Royal College of Surgeons of England, being the custodian of the Hunterian Museum of the College, which may be described as the National Medical Museum of the Empire, offered its services to collect and prepare the medical material for the whole British Expeditionary Force. In national spirit the College came to the assistance of Canada and the other Dominions, and offered to receive and care for material collected by their medical units—to be, in fact, the clearing-house in which this material could be collected, and later allocated to the rightful possessors. Thus all pathological and other material from Canadian hospitals, whether overseas or in Great Britain, finds its way to Lincoln's Inn Fields, and has been reviewed in Professor Arthur Keith's annual report which has recently appeared. Already a large amount of this material has been sent to Canada, and at the notable medical gathering in May at Hamilton, Ontario, to which we have already called attention (THE LANCET, July 27th, p. 120), when five of the most important medical associations of Canada met together, one of the outstanding features was the exhibition of specimens thus sent over. At the concluding session of the congress one of the main resolutions passed was "that whereas these war specimens have aroused great interest both among military surgeons, civil practitioners, and the general public visiting the Congress, and since their further collection and their adequate preservation and development into a great Canadian War Museum must be of the utmost value in the education of the Canadian Army Medical Corps at the present time, as well as a fitting memorial to future generations of the sacrifices of our troops, be it resolved that a copy of this statement of appreciation be sent to Surgeon-General Fotheringham, D.G.M.S., with the recommendation that it be forwarded to the Minister overseas, as an expression of the warm support which the development of a Canadian Army Medical Corps Museum received from the members of the Canadian Medical Association." Sir Robert Borden's visit to the Royal College of Surgeons of England, on the afternoon of Wednesday, August 14th, was thus an act of appreciation on his part of the services rendered to Canada by the English College, and was the formal opening of the exhibition now being held there. This exhibition is not the sum total of Canadian war specimens, but includes some of the more interesting of the specimens. Among these may be mentioned wax and plaster casts of war injuries, made at Orpington by Major F. Lessore and his staff; bird's-eye views and drawings by Captain Mathews, C.A.M.C., a well-known Canadian artist, of the different buildings in Great Britain and overseas occupied as Canadian war hospitals; pathological specimens, photographs, and X rays of war wounds and diseases. The whole affords a striking idea of the human interest there is in the records of the sufferings undergone by the soldiers, and the endeavours made by the medical services to counteract those sufferings.

IN connexion with the scheme for the employment of officers and men invalided from the Navy and Army it has been suggested that in cases of shell shock, gas poisoning, and general breakdown such men as contemplate life in the Far Eastern Colonies and dependencies would be well advised first of all to submit themselves for examination by some medical expert in tropical disease.

THE King of Italy, on the recommendation of the Italian authorities, has conferred the following Honours on the British members of the Royal Italian Military Medical Commission, in recognition of the voluntary and valuable services which, in conjunction with their Italian colleagues, they have rendered in examining the Italian recruits: T. Vincent Dickinson, M.D., Officer of the Order of the Crown of Italy; James Donelan, M.D., Officer of the Order of the Crown of Italy; Andrew Morton, M.D., Cavalier of the Order of the Crown of Italy; Andrew Currie, M.D., Cavalier of the Order of the Crown of Italy. The Italian authorities take the opportunity of cordially thanking the British and Italian members of the Commission and the President, Dr. Commendatore Melandri, for the care and zeal displayed in the work.

A MONTHLY RECORD OF ATMOSPHERIC POLLUTION.

METEOROLOGICAL OFFICE: ADVISORY COMMITTEE ON ATMOSPHERIC POLLUTION: SUMMARY OF REPORTS FOR THE MONTHS ENDING *Dec. 31st, 1917.* *Jan. 31st, 1918.*

Place.	Rainfall in millimetres.	Metric tons of deposit per square kilometre. Insoluble matter. Tar.	Carbonaceous other than tar	Ash.	Soluble matter. Loss on ignition	Ash.	Total solids.	Included in soluble matter. Sulphate as (SO_3).	Chlorine (Cl).	Ammonia (NH_3).
ENGLAND.										
Leicester	21	0·05	1·93	2·34	1·09	2·81	8·23	1·63	0·28	0·10
London—										
Meteorological Office	27	0·10	2·43	3·59	1·42	3·56	11·10	1·27	0·61	0·04
Embankment Gardens*	—	—	—	—	—	—	—	—	—	—
Finsbury Park ...	24	0·20	1·32	5·72	2·97	6·58	16·79	3·85	0·58	0·05
Ravenscourt Park ...	22	Nil.	0·30	0·47	1·32	2·66	4·75	1·67	0·30	0·09
Southwark Park* ...	—	—	—	—	—	—	—	—	—	—
Wandsworth Com.* ...	—	—	—	—	—	—	—	—	—	—
Golden Lane ...	25	0·11	2·70	4·15	1·29	4·37	12·62	1·83	0·96	0·15
Malvern	24	Nil.	0·04	0·10	1·15	2·03	3·34	0·82	0·41	0·01
Manchester—										
Queen's Park ...	55	—	—	1·04	—	—	4·20	—	—	—
School of Technology	30	—	—	1·50	—	—	5·20	—	—	—
Newcastle-on-Tyne	56	0·22	4·34	10·47	2·37	3·39	20·80	1·82	0·88	0·09
Rochdale	—	—	—	—	—	—	35·69	—	—	—
St. Helens	53	0·46	2·85	3·38	2·65	5·82	15·16	3·16	1·80	0·26
SCOTLAND.										
Coatbridge	43	0·10	1·52	4·35	2·98	5·57	14·52	2·75	0·41	0·12
Glasgow—										
Alexandra Park...	53	0·14	2·15	4·28	1·41	3·84	11·82	2·27	0·24	0·13
Bellahouston Park†	67	0·43	2·46	71·48	2·29	3·63	80·29	2·82	0·29	0·05
Blythswood-sq. ...	73	0·11	2·18	4·31	0·29	6·25	13 14	3·35	0·38	0·18
Botanic Gardens	85	0·48	0·35	3·91	2·74	6·93	14·41	4·83	0·23	0·10
Richmond Park ...	65	0·49	2·41	5·21	1·56	3·90	13·57	3·04	0·20	0·19
Ruchill Park ...	77	0·30	1·58	3·40	0·77	8·06	14·11	2·76	0 21	0·11
South Side Park.	73	0·14	1·59	3·57	0·65	4·60	10·55	2·25	0·16	0 11
Tollcross Park ...	68	0·08	2·10	5·75	1·72	5·26	14·91	3·55	0·21	0·16
Victoria Park† ...	76	0·02	2·49	17·01	3·04	5·77	28·33	3·23	0·25	0·14

Place.	Rainfall in millimetres.	Metric tons of deposit per square kilometre. Insoluble matter. Tar.	Carbonaceous other than tar	Ash.	Soluble matter. Loss on ignition	Ash.	Total solids.	Included in soluble matter. Sulphate as (SO_3).	Chlorine (Cl).	Ammonia (NH_3).
ENGLAND.										
Leicester...	15	0·52	3·73	6 00	0·74	4·96	15·96	3·04	0·19	0·06
London—										
Meteorological Office...	69	0·27	3·96	6 43	1·16	5·85	17·67	2·19	0·98	0·10
Embankment Gardens	62	0·06	0 76	1·78	6·84	10·30	19·73	5·83	1·07	0 36
Finsbury Park ...	73	0·09	1·52	5·68	2·42	4·45	14·16	3 18	0·65	0·10
Ravenscourt Park	34	0·10	2·04	7·12	1·01	1·52	11·79	1·20	0·26	0 07
Southwark Park...	44	0·06	0 50	2·81	2·64	4·20	10·22	2·65	0·70	0·08
Wandsworth Com.	21	Nil.	0 10	0·31	0 79	1·42	2·61	0·94	0·25	0·03
Golden Lane ...	71	0·19	1·39	7·03	1·70	5·96	16·27	2·39	1 21	0·20
Malvern	69	Nil.	0·05	0·07	0·69	2 06	2·90	0·79	0·28	0 01
Manchester—										
Queen's Park ...	37	—	—	—	—	—	4·90	—	—	—
School of Technology	30	—	—	—	—	—	11·40	—	—	—
Newcastle-on-Tyne	58	0·39	3·26	4·56	2·69	3·62	14·52	1·78	0·83	0·08
Rochdale	—	—	—	—	—	—	—	—	—	—
St. Helens	74	0·67	3·37	5·32	2·85	10·47	22·68	5·68	2·29	0·56
SCOTLAND.										
Coatbridge	54	0·12	2·60	5·02	3·87	6·78	18·39	4·06	0·39	0·23
Glasgow—										
Alexandra Park...	86	0·04	3·67	6·06	2·41	5·66	17·84	3·40	0·26	0·25
Bellahouston Park	110	0·06	1·29	2·24	4·85	6·48	14·91	4·71	0·19	0·19
Blythswood-sq.§..	9	0·06	1·59	4·22	0·85	1·23	7·96	0·21	0·03	0·04
Botanic Gardens‖	—	—	—	—	—	—	—	—	—	—
Richmond Park...	111	0·09	4·82	11·56	4·21	8·65	29·33	4·85	0·18	0·56
Ruchill Park‖ ...	—	—	—	—	—	—	—	—	—	—
South Side Park..	119	0·33	5·57	6·00	1·43	6·43	19·76	3·76	0·15	0·23
Tollcross Park‖ ...	—	—	—	—	—	—	—	—	—	—
Victoria Park ...	112	0·02	1·57	3·66	5·66	6·87	17·78	4·52	0·16	0·33

* Bottle broken. † Sand present with suspended solids. ‡ Result lost through accident.
§ Total content of gauge as received in chemical laboratory. ‖ Carboy broken during the month.

"Tar" includes all matter insoluble in water but soluble in CS_2. "Carbonaceous" includes all combustible matter insoluble in water and in CS_2. "Insoluble ash" includes all earthy matter, fuel, ash, &c. One metric ton per sq. kilometre is equivalent to: (*a*) Approx. 9lb. per acre; (*b*) 2·56 English tons per sq. mile; (*c*) 1 g. per sq. metre; (*d*) 1/1000 mm. of rainfall.

The personnel of public health authorities concerned in the supervision of these examinations and of the analytical work involved remains the same as published in previous tables. The analyses of the rain and deposit caught in the gauge at the Meteorological Office are made in THE LANCET Laboratory.

REPORT FOR 1917 OF THE COUNTY M.O.H. AND SCHOOL M.O. OF LONDON.

PART II.—EDUCATION.

Medical inspection.—In a previous report Dr. Hamer adduced the improvement then apparent in the results of medical inspection as evidence that the solicitous care for child welfare which had distinguished the history of recent years still remained undiminished by the circumstances of war. This improvement has been maintained ever since. Moreover, a further indication of the favourable economic conditions obtaining in 1917 is afforded by the fact that the number of necessitous children receiving free meals continued to decline below the already remarkably low figures of the preceding year. It is with profound satisfaction that the medical officer records these facts, showing as they do that the horrors of war had to this extent failed to affect injuriously the rising generation. Nevertheless, we learn that certain unfavourable conditions apparently inseparable from warfare still remain unredressed. Prominent among these is the difficulty of obtaining clean and wholesome milk for the feeding of necessitous infants. On analysis half the samples of such milk already examined have been found to contain added water. Apart from a few small and localised outbreaks of dysentery in districts north of the Thames, two occurrences prejudicial to health were reported as directly attributable to the war. In the first place, there was marked increase in the prevalence of scabies, doubtless introduced by soldiers returned from abroad. Secondly, a falling off was noticed in the results of treatment of children's diseases. This was caused partly by the withdrawal of hospital facilities and partly by the failure of arrangements for "following up." In some degree that failure was due to the employment of nursing mothers away from home, but mainly to falling off in the supply of voluntary care workers.

Effects of Economic Conditions.

The annual individual examination of a quarter of a million children in London affords a reliable measure of the social condition of the community, with which there had previously been nothing to compare. In regard to personal cleanliness the maintenance of the *status quo ante* has been fully achieved, but only by constant vigilance on the part of the nurses. This is remarkable, in face of the persistent introduction of parasitic infection and of the increased employment of married women. While the proportion of children ill-fed and insufficiently clothed has remained low, there has been a slight reduction in that of children well dressed and shod, as contrasted with those only fairly well provided for. It would appear that economic considerations have prevailed here, and that parents are now beginning to spend more on actual necessaries and less on finery of apparel than in the earlier years of war. Scrutiny of the figures for nutrition reveals the indirect influence of war conditions on the physical condition of the children. The progressive improvement in their appearance has been maintained throughout the last two years; the number of poorly nourished children having been only half as great as in pre-war times. The returns for the third term of last year were, in this regard, the best hitherto attained. On the other hand, the proportion of children returned as of "good nutrition," as opposed to "only fair nutrition," has shown some diminution. Coöperation has been established with several infant-welfare centres. At six of these arrangements have been made by which the same premises are used for the purposes of school treatment and infant welfare, to the mutual benefit of all concerned.

The open-air schools at Birley House and Shooter's Hill are still continued. The improvement in the mental as well as the physical state of the pupils has been well marked; the benefit to debilitated and anæmic children accruing from open-air education, supplemented by generous feeding,

being especially obvious. The majority of those in attendance are children in whom the onset of tuberculosis was feared, several of them having returned from sanatoriums with the disease in an arrested state. Further coördination of school work with that of the tuberculosis dispensaries has been effected with advantage to the children. The Kensal House School for tuberculous patients has been continued and some of the playground classes have been held in the public parks. The adequacy and quality of the food supplied to the schools has been attended to, the report containing results of the analysis of samples of meals supplied to the pupils. Save as regards milk (as previously mentioned) there has been found no cause for complaint.

Personal hygiene.—There are now 25 cleansing stations in London, at most of which treatment is extended by the inclusion of scabies. More than 30,000 baths were given to 6940 children suffering from scabies. This large increase on previous records was, doubtless, caused by infection introduced into the homes mainly from abroad. During the year 799,000 children were examined by the nurses, and in 19,500 cases verminous conditions were found. 8500 of the children were cleansed by parents and the remainder by the borough councils.

Verminous conditions and infectious disease.—The year under notice was remarkable for the extent to which attack was made on problems arising out of verminous conditions. How great is the improvement in this respect within the last 30 years will be apparent to those having had experience of London school children extending over that period. But unfortunately the campaign against vermin has not until lately excited general enthusiasm, and consequently the Council's nurses who have endeavoured to combat the prevailing indifference respecting cleanliness have had a difficult and sometimes unpopular duty to perform. It is now, however, realised that the banishment of plague in the seventeenth century was probably effected by attacking the prevalence of vermin, and that the practical extinction of typhus and relapsing fevers in this country within the last half-century has been accomplished mainly by improving the conditions of the poor as regards infection with body lice. Of late years study of plague in India and of the part played by insect parasites has greatly emphasised the importance of fleas, and it may well be that it is now necessary to look nearer home. Dr. Hamer refers to the inquiries recently made in London respecting the relationship between the "flea curve" and the curve of scarlet fever; he claims that the case in favour of a flea hypothesis of scarlet fever causation is one that calls for close examination. The amount of misery and discomfort caused by flea-bites among children has incidentally been revealed, but it is not possible to allow the question to rest at this point. The demonstration of the association of vermin with the spread of infectious disease has done more than had ever before been accomplished in arousing serious interest on the subject. The fact that during the past year one London elementary school-girl out of three has been found to have "nits" in her hair is a condition of things for which the medical officer is determined that a remedy must be found. The spread of verminous conditions among soldiers and the dissemination of scabies throughout the country have enforced the moral, and it is to be hoped that the attempt to cope with this national danger will meet with general support.

Among the commoner diseases of school life there was less prevalence than usual. Scarlet fever was reported in 6000 instances and diphtheria in 8000. Measles and German measles occurred among school children in 27,000 instances. Whooping-cough was less prevalent in 1917 than in any year of the preceding five.

THE LATE MR. WILLIAM FINGLAND, L.R.C.P., L.R.C.S. EDIN.—Mr. W. Fingland, honorary consulting anæsthetist to the Liverpool Royal Infirmary, died on August 1st from cardiac failure at his residence, Rodney-street, Liverpool, at the age of 66 years. Educated at Liverpool, he qualified with the licentiateship of the Royal Colleges of Edinburgh in 1891 and settled in practice at Liverpool, where for 25 years he was on the staff of the Royal Infirmary. He was vice-president of the section of anæsthetics when the British Medical Association held its meeting in Liverpool in 1912. Mr. Fingland leaves a widow and a daughter.

Correspondence.

"Audi alteram partem."

NASO-PHARYNGEAL INFECTION AT THE ONSET OF CEREBRO-SPINAL FEVER.

To the Editor of THE LANCET.

SIR,—Lieutenant-Colonel M. H. Gordon, R.A.M.C., in his letter to THE LANCET of Nov. 10th, 1917, and in his report to the Medical Research Committee, 1917, states that "every case of cerebro-spinal fever was in reality an instance of a carrier developing the disease," and that the meningococcus was invariably to be found in the naso-pharyngeal secretion at the onset of the disease. I wish to mention the following findings as contravening the above statements, of which findings Colonel Gordon is already aware, both from my reports and from conversations I have had with him, but to which he does not allude either in his letter or in his report.

In the spring of 1916, whilst employed in coördinating the work of investigation and prevention of the outbreak at X Garrison (pages 19 and 70 of above report), I paid particular attention to the question of infection in the naso-pharynx, and excluding some earlier cases, where the results could not be relied upon, a special record was made of the results of swabbing of all patients on admission to hospital. Forty-one cases were examined on admission, and the meningococcus could be demonstrated in 19 only. There was no greater degree of infection in those cases actually diagnosed as cerebro-spinal fever than in a group of other febrile patients suffering from influenzal or other complaints who were being admitted in epidemic numbers at the same time. The symptoms of many of these, however, approached in severity those actually diagnosed as cerebro-spinal fever, and I suggested in my report that they were in many cases really caused by the meningococcus.

The swabs were taken and examined by Captain C. Samut, R.A.M.C., professor of bacteriology, Malta University, and by Captain R. R. Armstrong, R.A.M.C., at that time attached to the Central Cerebro-spinal Laboratory.

I discussed these negative results with Colonel Gordon, who was inclined to query the diagnosis. Captain Samut had, however, demonstrated Gram-negative intracellular diplococci in the spinal fluids of all the diagnosed cases, nearly half of which yielded a successful culture and were agglutinated; and Colonel Gordon had agglutinated to type the meningococcus derived from five patients who failed to show the meningococcus in the naso-pharynx. Captains Trevor Davies and E. G. Pringle, who were in medical charge of the patients, and all of us concerned, were entirely of opinion that these cases were really cerebro-spinal fever. Colonel Gordon had not seen the patients.

In my report of this epidemic I gave further details, pointing to an absence of meningococci from the fauces of patients within a period of a few hours to a few days before the onset of symptoms, as follows:—

3	soldiers	were	negatived	within	24	hours	of onset.
2	,,	,,	,,	,,	2	days	,,
2	,,	,,	,,	,,	3	,,	,,
1	,,	,,	,,	,,	4–7	,,	,,
1	,,	,,	,,	,,	7	,,	,,

These soldiers were swabbed when isolated as contacts immediately after the diagnosis of a case, and they developed cerebro-spinal fever within the period stated. 520 carriers (contacts and non-contacts) were isolated for a long period at X; none of them developed any illness.

In April, 1917, I had an opportunity of confirming the negative findings of the year before whilst on sick leave from abroad, when I was temporarily attached to the Central Cerebro-spinal Laboratory. Captain Glover, R.A.M.C., in charge of investigation of cases in the London area, kindly swabbed for me four definite cases of cerebro-spinal fever which we visited together, and in only one of these could the meningococcus be grown afterwards in the Laboratory, although two of them were tried twice. On mentioning these results to Colonel Gordon, he had a tabular list prepared of the London cases during the previous couple of months. This list comprised 19 cases, 7 of which were negative and 6 positive in the naso-pharynx; the remainder had not apparently been tested, as no results are mentioned on the list. These cases were open to no doubt, and the meningococcus was isolated from the spine in each case.

Other observers have likewise noted the absence of meningococcus from the fauces of patients. In the Advisory Committee's report it is stated that "the meningococcus has

been much less commonly found in the naso-pharynx of actual sufferers than might have been expected." Gaskell states that only 25 per cent. are infected, and mentions Netter and others as finding infection in 60 per cent. of cases of the disease.

Colonel Gordon, however, considers that the meningococcus is always present, so much so that he and other officers working with him in the Central Laboratory regard its presence as practically essential to a diagnosis. He states as follows (Medical Research Committee Report, page 19):—

"The constant presence of the meningococcus in the naso-pharyngeal secretion at the earliest stage of the disease is a point of considerable practical importance, for by the simple procedure of taking a naso-pharyngeal swab and plating the secretion on the spot cases of cerebro-spinal fever can be recognised with practical certainty within 48 hours of being called to see them. The evidence yielded by the naso-pharyngeal swab has been found by Captain Flack to be of cardinal importance in facilitating the diagnosis, particularly in early cases, in cases where the symptoms are atypical, or where the spinal fluid fails to yield a positive culture." (See also Captain Flack's record on diagnosis on page 41 of the report.)

The diagnostic application thus made of the invariable presence of the meningococcus in the naso-pharyngeal secretion of the patient at the onset of cerebro-spinal fever naturally explains why all the cases investigated are reported as harbouring the meningococcus, but this method of diagnosis does not appear logical inasmuch as the symptoms cannot be reasonably described as atypical when the physician in charge has already submitted his patient to lumbar puncture, and also because it is a well-known fact that the meningococcus as often as not fails to grow when cultured; on the other hand, there are not many bacteriologists who would place reliance in diagnosis on the result of plating the secretion from the naso-pharynx alone.

In May, 1916, I presented a report on cerebro-spinal fever based on the epidemics at X and other garrisons. Last year I applied to publish a further report on the same lines dealing with the epidemiology of the disease. I was then ordered on active service, since when I have not heard news of its publication.

In these reports arguments and statistics were brought forward to emphasise the following points:—

1. The existence of many mild and usually unrecognised cases of cerebro-spinal fever.
2. The prime importance of these mild cases on the spread of the infection.
3. The impracticability of controlling the disease by isolation of carriers.
4. The importance of recognition and of isolation of mild and severe cases, and of the provision of free ventilation during the period of seasonal prevalence as the most effective means of prevention.

I am, Sir, yours faithfully,

J. DORGAN,
Major, R.A.M.C.

Feb. 25th, 1918.

Major Dorgan's letter has taken some months to reach THE LANCET, and to avoid further delay we have submitted it to Colonel Gordon, who replies through us as follows:—

SIR,—I would remind Major Dorgan, in the first place, that pathological and bacteriological tests are far more valuable in their positive than in their negative aspect. This is well known in the case of diphtheria and tubercle, where failure to find the specific micro-organism in a single examination of material from a clinically suspicious case is recognised as being by no means conclusive. Still more does it hold with regard to the meningococcus, since the technical difficulty of isolating and identifying this highly delicate micro-organism in the naso-pharyngeal secretion is considerably greater, and success requires special training, experience, and sometimes considerable perseverance as well.

The history of the literature on the subject of the presence of the meningococcus in the naso-pharyngeal secretion of patients suffering from cerebro-spinal fever illustrates this difficulty in a striking manner, notably the experience of v. Lingelsheim, who during the Silesian outbreak at first found the meningococcus in the naso-pharyngeal secretion of only 182 out of 787 cases, or 23 per cent.; but later, when as the result of riper experience he made his cultures at the bedside and incubated them at once, he succeeded in detecting this micro-organism in the naso-pharyngeal secretion of 46 out of 49 patients, or 94 per cent.

An important point to be borne in mind in this relation is the tendency which the meningococcus shows to disappear from the naso-pharyngeal secretion of the patient as the disease progresses.

Lingelsheim's findings were as follows:—

Day of disease.	No. of specimens examined.	No. positive.	Per cent. positive.
First to fifth day	156	104	66·6
Sixth to tenth day	57	14	24·5
Eleventh to twentieth day ...	62	7	11·2
Twenty-first day and after ...	115	5	4·39

Similar results have been reported by Goodwin and v. Sholly and by Netter and Debré. If in addition it is realised that in a proportion of cases of c.s. fever among troops the patient when first seen by the bacteriologist is in a condition of delirium or coma, as the result of which his naso-pharynx is by no means easy to get at, and the secretion there perhaps either dried up or grossly contaminated by other bacteria, failure to demonstrate the meningococcus in this secretion can be readily understood. Nevertheless, the positive findings already referred to, and the observations made by Captain Martin Flack, R.A.M.C., when in charge of the London District C.S. Fever Laboratory, by Major F. W. Andrewes and Dr. R. G. Canti at St. Bartholomew's, and by others, indicate, in my opinion, that the meningococcus is practically always there at the onset, and is to be found if the investigator has the experience and equipment and is able to devote the necessary time to searching for it. In this relation it may be mentioned that Captain Flack found that it is always advisable to take at least two swabs when examining the naso-pharyngeal secretion of a patient for the meningococcus.

With regard to a considerable proportion of the cases of suspected cerebro-spinal fever at X, to which Major Dorgan refers, I must say that, notwithstanding the careful investigations of Captain Samut, the evidence that they were really infected by the meningococcus is far from complete. The mere presence of a very few diplococci found free or lying in, or on, leucocytes in films of the deposit of a clear cerebro-spinal fluid from a case of transient illness can hardly be accepted *per se* as conclusive bacteriological evidence of cerebro-spinal fever. With every care, the chances of introducing a few sterilised diplococci from the skin of the patient's back must be borne in mind. The positive bacteriological evidence necessary to establish Major Dorgan's ingenious hypothesis that these were really cases of c.s. fever has still to be provided, and even if some of them were abortive cases of that disease, the part they play in its spread has still to be proved.

I am, Sir, yours faithfully,

M. H. GORDON,
Lieutenant-Colonel, R.A.M.C.

Central C.S.F. Laboratory, 12, Caxton-street, S.W. 1,
August 9th, 1918.

WAR DEAFNESS.

To the Editor of THE LANCET.

SIR,—No science has learnt more lessons from the war than medicine. Our knowledge of functional nervous disorders has, for example, greatly increased owing to the frequency of these disorders in soldiers. Old methods of diagnosis have had to be revised, and the results of treatment have steadily improved. The danger of relying on pre-war ideas has nowhere been more obvious, and it has already become necessary to discard many of the views which were prevalent early in the war. Thus, if we relied on the papers published since 1915 by Meige and by Oppenheim, distinguished neurologists as they are, we should still regard the tremors which are so common in shell shock as almost incurable, whereas we now know they respond to psychotherapy almost as rapidly as the more obviously hysterical symptoms. In the same way, if we had accepted the views of Babinski and Froment on "reflex" paralysis and spasm, we should have failed to cure the very common paralyses and contractures of the hand and foot, which have been proved by the rapid recovery under psychotherapy to be hysterical and not reflex in origin. Since the translation of their book the views of Babinski and Froment on reflex nervous disorders have unfortunately been widely accepted in England, as, for example, by Major Judson S. Bury in his paper in THE LANCET of July 27th, although Captains J. L. M. Symns, W. R. Reynell, and A. W. Gill, Lieutenant S. H. Wilkinson, and I have not yet failed to cure by psycho-

therapy a single one among a very large number of cases presenting all the signs and symptoms of so-called reflex paralysis or contracture.

It is unfortunate that two distinguished aural surgeons, Dr. P. McBride and Dr. A. Logan Turner, should have been led by their pre-war experience to write such a pessimistic article on "War Deafness" as that which appeared in THE LANCET of July 20th. If we had believed, like them, that the majority of cases of concussion-deafness are organic in origin, and are consequently incurable, or at the best can only improve slightly, we should never have attempted psychotherapy, whereas in our experience many are completely curable by re-education and suggestion, and very few are incapable of some improvement by the methods which have been devised as a result of war experience. Our results are identical with those obtained by Gordon Wilson and Yealland in England and Roussy and Lhermitte in France. My own "opportunities of studying ear diseases" before the war were fortunately nil, and I was therefore able to face the problem of war deafness with an unprejudiced mind. As it is obvious that suggestion cannot prevent the occurrence of nystagmus on testing the vestibular reaction by rotation or cold water, the loss of this reaction after concussion proves that some organic change has occurred in the internal ear, and that any deafness present is likely to be at least in part organic in origin. On the other hand, total deafness caused by concussion, if associated with a normal reaction, is probably hysterical and due to suggestion, as it is very unlikely that the cochlea should be so damaged that complete deafness results without the adjacent semicircular canals being affected. In cases of incomplete deafness the sign is of less value, but we have acted on the principle that whereas *all* cases of deafness following concussion should be treated by psychotherapy—in the hope that it may be at least in part functional, like the frequently associated mutism, blindness, tremor, or paralysis—we expect to obtain the best results, and we persevere with our treatment for a longer period, when the reactions are normal and definite evidence of organic injury is absent. Our results certainly justify our methods, as we have produced more or less improvement in almost every case and have cured men who have been totally deaf for many months, sometimes after they had been told by distinguished aurists that they could never recover from their "nerve deafness." In contrast to this, Dr. McBride and Dr. Logan Turner appear to have only seen improvement in one case, and in that case it appears to have occurred spontaneously, as it is hardly conceivable that any good could have resulted from the use of pilocarpin, which certainly stimulates the secretion of glands, but has never been proved to stimulate the special senses.

Dr. McBride and Dr. Logan Turner believe that "very grave injustice might be done" by regarding deafness as probably hysterical when the vestibular reactions are normal; we, on the contrary, regard it as most unfair to one's soldier patients to be so prejudiced by pre-war experience that the possibility of functional origin is not considered, the patient being consequently deprived of the chance of cure by psychotherapy. I should regard Case 2 in Group C and Case 1 in Group D as almost certainly hysterical, and curable within a few days and possibly within a few minutes, whereas the authors state that because there is a history of head injury they could exclude all question of functional trouble "with reasonable certainty." The patients therefore remained untreated and will doubtless remain deaf for the rest of their lives, as being told by a distinguished specialist that the trouble is incurable, their disability, which was originally caused by auto-suggestion, would become perpetuated by hetero-suggestion, and the chance of cure by psychotherapy would be enormously reduced.

I am, Sir, yours faithfully,

ARTHUR F. HURST.

Seale Hayne Military Hospital, Newton Abbot, July 28th, 1918.

TREATMENT OF WAR NEUROSES.

To the Editor of THE LANCET.

SIR,—Upon the rapidity of their results as to the removal of hysterical symptoms and on the sensible form of suggestion which they have evolved, Major A. F. Hurst and Captain J. L. M. Symns are to be congratulated. It would greatly enhance the value of their contribution to THE LANCET of August 3rd if they could speak with more definition as to the permanence of the "cures" they mention, and would publish statistics as to the state of their patients six months and one year after "cure." The fact that there are many thousands (30 or 40 I believe) of pensioners, many of them drawing full pensions, under the control of the Special Medical Board, accentuates the necessity of not only removing hysterical symptoms, but of also confirming the cure of these men straight away, before any relapse has occurred.

I have suggested to the medical authorities of the Navy and Army that many, if not all, men who have suffered from war neuroses should be sent, after treatment at a neurological centre, but before discharge to the country, under the auspices of the Country Host Institution, so that they may do outdoor work in the fields and gardens, in conditions half-way between the complete control of a hospital and the sentimentality, worry, and responsibility of their own homes. The Director-General of the Medical Department of the Navy at once accepted the suggestion and some 30 undischarged sailors have already been sent off and are making very satisfactory progress; the Director-General, A.M.S., has not yet informed me whether he intends to fall in with my proposal or not.

I rejoice to find that Major Hurst and Captain Symns agree with my very strong conviction that none of these functional nerve cases should be treated in London, subject as it is to air-raids; all hospitals dealing with such cases should at once be transferred to the country.

The ideal treatment for war hysteria is, I hold: (1) removal of symptoms, &c., by such methods as Major Hurst and Captain Symns use; (2) a three months' course of Country Host convalescent treatment to strengthen the men, re-educate them in the habit of work, and thus confirm the cure effected at the neurological centre. Neurasthenic (nervous debility) cases can with advantage be sent direct to the country for convalescence, thus obviating the necessity of their occupying valuable hospital accommodation.

I am, Sir, yours faithfully,

August 3rd, 1918. THOMAS LUMSDEN.

INFANT MORTALITY AND MOTHERCRAFT.

To the Editor of THE LANCET.

SIR,—One would hardly imagine, on reading Dr. W. A. Brend's letter published in your issue of July 27th in reply to my article of July 6th, that the main point at issue between us is (to quote from his own report)—

"That the excess (of infant mortality) in industrial towns, the centres of large cities, and mining areas is due to some factor or factors, of which possibly *the most important is a polluted state of the atmosphere.*"

He says it probably is; I say it is not. Instead of addressing himself to this issue he concludes his letter of the 27th by writing:—

"I am of opinion that the main cause of excessive infant deaths, as of tuberculosis, malnutrition of school children, and other evils, is overcrowding of houses, congestion of streets, and the absence of large and sufficient open spaces."

Cannot Dr. Brend see that these things mean far more than a polluted atmosphere, and that, under such conditions, mothercraft, and all that it implies, becomes of paramount importance?

He is wrong in stating that my main criticisms of his report are two in number—viz., that he has endeavoured to eliminate poverty and under-estimated the value of mothercraft! My article was an attempt to do much more than that; it was an attack all along the line against his method of proof by a process of exclusion—a process which, I hold, was singularly illogical and unconvincing, and in certain directions, to be candid, not quite fair or judicial.

If Dr. Brend will get into contact with the working-class inhabitants of any large town, he will soon find for himself that far the larger amount of the excess infant mortality in that town does occur amongst the children of the unskilled and casual workers and others of the same social station. That they are living under the worst housing conditions I grant, but it is also amongst that class that the worst housewifery and mothercraft exists.

He further says that nowhere has he suggested that maternity and infant welfare centres should be incontinently scrapped. I never said he had; but that would be the

logical outcome of his report, if accepted, and I suggested that that was the question we who are interested in these institutions must put to ourselves.

I am obliged to Dr. Ronald Carter for his support. From his own long and practical experience he appreciates, I know, the real difficulty of compiling thoroughly satisfactory mortality tables for consultation infants. It could be more satisfactorily done by medical officers of health than by medical officers to consultations, most of whom in times past have not been directly connected with Public Health departments, nor, therefore, had the facilities for the extended inquiry which such tables imply.

I hope, however, in due time to be in a position to publish some statistics dealing with this point.

I am, Sir, yours faithfully,

H. W. POOLER, M.B.,
Medical Superintendent, Derbyshire County Infant Welfare Centres.

Stonebroom, Alfreton, August 11th, 1918.

THE LATE MR. W. MORLEY WILLIS, OF NOTTINGHAM.

To the Editor of THE LANCET.

SIR,—We should like to record the loss to British surgery in the death of that brilliant young surgeon, Mr. W. Morley Willis, F.R.C.S. Eng. Clear-headed, indefatigable, and self-confident, he was bound, if he had lived, to take a place in the first rank of his profession; his death seems to have escaped public notice.

He was a native of Bristol and was educated at Taunton Grammar School. A business career had been arranged for him, and for some time to meet the wishes of his relatives he made an effort in this direction, although from boyhood his ambition was to be a surgeon. But with his twenty-first year he brushed aside all other considerations and commenced the study of medicine at University College, Bristol. When he qualified in 1895 he continued his surgical studies at St. Bartholomew's Hospital, London, taking the Fellowship of the Royal College of Surgeons of England in the following year.

Commencing practice in Nottingham in 1897, he joined the surgical staff of the Children's Hospital, and in 1901 he was appointed assistant surgeon to the Nottingham General Hospital. Soon after this he abandoned general practice and confined himself entirely to surgery. In addition to the public work which these appointments entailed, Mr. Willis's heavy duties in connexion with various military hospitals made his life latterly a very full one. Two months ago, while well, cheerful, and full of work and vigour, he was struck down with fatal illness, which he faced with extraordinary courage and pluck. Mr. Willis was not only a brilliant surgeon and skilful diagnostician, but a kindly and courteous colleague, always ready to give help and advice in difficult cases and eager to assist the younger men in any way he could. He has been greatly missed by his colleagues and by his patients, who loved him for his cheery optimism and charming manner. Mr. Willis leaves a widow and two daughters.—We are, Sir, yours faithfully,

JOHN MACKIE, L.R.C.P. Edin.,
H. BELL TAWSE, F.R.C.S. Eng.,
Assistant Surgeon to the Nottingham General Hospital.

August 9th, 1918.

COLONIES IN THE TREATMENT OF PULMONARY TUBERCULOSIS.

To the Editor of THE LANCET.

SIR,—I note in your issue for August 3rd two articles on the above subject. To me it appears the eternal mistake of not knowing the beginnings of tuberculosis is being made. In my experience very few early cases of pulmonary tuberculosis require more than three months in a sanatorium, and those that require more would seldom be better for being made to work. Colonies are only suitable for chronic cases—that is, cases which have not been treated properly when exhibiting symptoms of activity. Whether the expense involved in treating cases of this kind for prolonged periods is justified is more than open to doubt. That it is impossible to treat tuberculosis in the early stage at dispensaries would seem obvious to anyone who has had to deal with this disease; some might at great risk to themselves be treated at home, but never by walking about. That early cases require as long as 18 months must also usually be an error, and it seems probable that the truth must lie between these two extremes. I should like to suggest to physicians to take a previous and careful history of the health of all the cases they regard as early, and in the light of the new facts re-grade their cases and try to avoid allowing their future patients to become chronic.

I am, Sir, yours faithfully,

EDWARD E. PREST.

Ayrshire Sanatorium, New Cumnock, August 7th, 1918.

THE SERVICES.

ROYAL NAVAL MEDICAL SERVICE.

Surgeons (Reserve) to be Staff Surgeons (Reserve): J. P. Berry, C. F. Bainbridge, S. N. McBean. To be temporary Surgeons: W. E. M. Wardill, V. T. Smith, S. C. Mitchell, S. Acheson.

The following appointments have been made:—Surgeons (temporary): H. Whyte to *Centurion*: B. F. Niblock to Chatham Hospital; J. D. Arthur to Plymouth Hospital; and W. Edgar to Haslar Hospital.

ARMY MEDICAL SERVICE.

The undermentioned temporary appointments are made at the War Office: Dep. Asst. Dir.-Gens.: Capt. E. F. W. Mackenzie, and to be temporary Major whilst so employed; Temp. Major S. Fleming.

ROYAL ARMY MEDICAL CORPS.

T. B. Rhodes, late temp. Major, is granted the honorary rank of Major.

M. A. Collins, late temp. Capt., to be temporary Lieutenant-Colonel whilst serving at the Ewell (County of London) War Hospital.

Temporary Captains relinquishing their commissions and are granted the honorary rank of Captain: J. Glaister, on account of ill-health contracted on active service; L. M. Webber, on account of ill-health caused by wounds.

Temporary Lieutenants to be temporary Captains: R. T. St. J. Brooks, D. G. Halsted, H. E. Gray, J. L. McCann, E. H. R. Harries, W. W. Walker, A. M. Hewat, (Acting Major) D. Burrows, J. A. Loughridge, J. A. Clarke, H. M. Raven, R. M. Menzies, W. A. Mahon, R. McN. Wilson, G. M. Coope, A. F. Sanderson, A. M. Jones, A. Rhodes, M. Hocken, N. K. Foster, J. C. Middleton, A. Allison, C. W. Ewing, R. Wade, E. D. W. Reid, C. R. Wilkins, K. H. Bennett, F. H. Flack, S. S. M. Wood, (Acting Major) R. G. Oram, G. J. Eady, D. Martin, H. Sinson, J. Hunter, A. Brownlie, J. H. Thompson, J. E. R. McDonagh, L. G. Leonard, D. A. Stewart, C. Garner, J. A. Brown, T. N. Wilthew, W. Murray, F. Butler, A. White, J. D. Kenyon, W. J. McFeat, T. D. Graham, H. J. van Praagh, R. N. Berman, A. Bradshaw, A. G. Craib, D. M. McGillivray, A. D. Yule, W. Gorrie, W. Boyd, T. W. Bayne, G. B. Charnock, R. Parry, G. B. Warburton, G. Newstead, W. Angus, J. K. Willis, F. B. O'Dowd, G. H. Mead, R. J. Reynolds, J. H. Sutcliffe, H. H. Jenkins, J. K. Hamilton, G. A. Rorie, A. S. Campbell, A. T. MacKenzie, S. Bree, S. G. Corner, D. A. Dewar, W. J. D. Bromley, A. Davies, R. Davidson, J. W. Simon, K. R. C. Hallowes, F. D. Parbury, S. L. O. Young, N. McC. Hutchison, F. D. Nicholson, W. L. English, E. Banks, A. S. M. Palmer, E. E. Cassady, L. D. Stamp, H. P. Dawson, A. M. Laurie, J. Donaldson, H. M. Reeve, M. F. Emrys-Jones, W. G. Riley, A. Morton, A. Barker, D. Purdie, J. F. Findlay, H. J. Beddow, R. L. Haines, E. F. Thomas, A. D. Crofts, H. J. Rae, G. C. B. Miéville, C. W. Hutt, R. S. Roper, P. W. Stewart, F. F. Laidlaw, A. V. Ledger, N. C. Rogers, H. Snape, K. S. Melvin, J. R. Cameron, T. Miller, A. Robertson, P. E. Carroll, M. T. D. McMurrich, A. Boyle, E. E. Atkin, J. Fletcher.

Temp. Hon. Lieut. A. H. Good to be temporary Honorary Captain.

Temp. Lieut. Michael J. MacCarthy relinquishes his commission.

TERRITORIAL FORCE.

Major (temp. Col.) A. Milne-Thomson relinquishes his temporary rank on vacating appointment as Assistant Director of Medical Services, and is restored to the establishment.

Major (Bt. Lieut.-Col.) T. D. Acland remains seconded whilst holding a temporary commission in the R.A.M.C.

Capt. A. N. McGregor is restored to the establishment.

Capt. J. Allan to be Major.

Capt. A. E. Woodhead is seconded for duty.

Capt. (acting Major) H. Henry relinquishes his acting rank.

Capt. A. McLean is seconded for duty with General Hospital.

R. B. Driscoll and S. Shaw, R.A.M.C., to be Lieutenants.

ROYAL AIR FORCE.

Medical Branch.—A. P. Woolright (late Captain, R.A.M.C.) and W. G. West are granted temporary commissions as Captains.

P. A. Dormer, R. W. Stephenson, G. F. H. Bloom, H. B. B. Greene are granted temporary commissions as Lieutenants.

Dental Branch.—W. C. Murray (late Dental Surgeon, R.N.V.R.) is granted a temporary commission as Captain and to be temporary Major whilst specially employed.

DEATH OF MR. B. G. DERRY, L.R.C.P., M.R.C.S.—Mr. Bartholomew Gidley Derry died on August 8th, at his residence at Bodmin, from the effects of a carriage accident. Mr. Derry, who was in his seventy-second year, had practised in Bodmin for 47 years. He had an extensive practice and he was medical officer of health for the Bodmin (Cornwall) urban district, surgeon to the Civil and Naval Prisons at Bodmin, and also held other appointments in which he earned high respect in Bodmin and the district. He leaves two sons and three daughters.

The War.

THE CASUALTY LIST.

THE following medical officers appear among the casualties announced since our last issue:—

Killed.

Capt. H. R. Partridge, M.C., R.A.M.C., qualified in London in November, 1914, and was attached to the 1st West Riding Field Ambulance in the early part of the following year. The award of the M.C. was recorded in THE LANCET of Oct. 28th, 1916, p. 770.

Reported Missing, now believed Killed.

Capt. W. H. Lloyd received his medical education at the Middlesex Hospital, and qualified in 1915. He became attached to the 3rd London General Hospital in August, 1915, and had been in France since June, 1916.

Died.

Surg. Prob. A. E. Beckwith, R.N.V.R.
Lieut. D. Taylor, R.A.M.C., qualified at Glasgow in July, 1917, and joined the R.A.M.C. quite recently.

Wounded.

Capt. R. Anderson, R.A.M.C., attached Grenadier Guards.
Capt. W. Baxter, R.A.M.C., attached Gordon Highlanders.
Major G. Stiell, R.A.M.C.
Col. T. W. Gibbard, C.B., K.H.S., R.A.M.C.
Capt. E. M. McCaffrey, Australian A.M.C.
Lieut. J. J. Hughes, R.A.M.C., attached N. Staffs Regt.
Capt. A. W. Ritchie, R.A.M.C.
Lieut. J. J. Pickles, R.A.M.C., attached W. Yorks Regt.

Previously reported Missing, now reported Prisoners in German Hands.

Capt. C. R. Crowther, R.A.M.C.
Capt. W. T. P. Meade-King, R.A.M.C.
Capt. G. F. P. Heathcote, M.C., R.A.M.C., attached East Lancs Regiment.
Capt. F. C. H. Bennett, R.A.M.C., attd. Northumberl'd Fus.
Lieut. A. Boyle, R.A.M.C.
Capt. M. S. Esler, R.A.M.C., attached Middlesex Regiment.
Capt. D. D. Gillespie, R.A.M.C.
Lieut. J. W. Jones, R.A.M.C., attached Rifle Brigade.
Lieut. A. M. McCormick, R.A.M.C., attd. Lincs Regiment.
Capt. C. Witts, M.C., R.A.M.C.

MENTIONED IN DESPATCHES.

The names of the following medical officers are mentioned in a despatch by Lieutenant-General Sir J. L. van Deventer, K.C.B., Commanding-in-Chief, British East Africa Force, for distinguished services during the operations from May 30th to December, 1917:—

Royal Army Medical Corps.—Lt.-Col. (acting Col.) F. E. Gunter, D.S.O.; Capt. W. Fraser; Capt. J. O. Garland; Temp. Lt. J. I. Greig; Capt. A. Hendry; Capt. H. F. Holmden; Temp. Capt. C. R. Howard; Lt.-Col R. H. Humphrey; Maj. G. J. Keane, D.S.O.; Temp. Capt. A. R. Lindsay; Temp. Capt. L. R. P. Marshall; Temp. Capt. J. Miller; Capt. W. Mitchell; Temp. Capt. M. T. Morgan, M.C.; Temp. Capt. A. Moxon; Temp. Capt. W. A. Ryan; Temp. Lt. R. Semple; Capt. E. A. Sutton, M.C.; Temp. Capt. W. B. Walker; Capt. G. A. Williams.

Royal Army Medical Corps (S.R.).—Capt. H. S. A. Alexander; Lt. J. F. C. Braine; Capt. S. J. V. Furlong; Capt. R. L. Impey, M.C.; Lt. R. Lloyd-Jones; Lt. F. C. Logan; Capt. W. A. McLennan; Capt. G. M. Roberts; Capt. A. R. Ross; Lt.-Col. J. McKie, D.S.O.

Indian Medical Service.—Lt.-Col. (Temp. Col.) W. W. Clemesha; Capt. F. W. Hay, attd. Inf.; Capt. T. A. Hughes; Maj. J. Husband; Capt. H. M. Inman; Capt. M. L. C. Irvine; Capt. G. G. Jolly; Capt. W. M. Lupton; Maj. P. L. O'Neill.

Indian Subordinate Medical Department.—2nd Cl. Asst. Surg. A. Bayley-de-Castro; Asst. Surg. (Hon. Lt.) J. A. F. Harvey; 1st Cl. Asst. Surg. E. D. Shave; 2nd Cl. Sub-Asst. Surg. Bhag Singh; 2nd Cl. Sub-Asst. Surg. Gordhandas Jethabhai Desai; 2nd Cl. Sub-Asst. Surg. Govind Ramchandra Upalekar; 2nd Cl. Sub Asst. Surg. Hari Singh; 1st Cl. Sub-Asst. Surg. Kashi-Nath Ram-Chandra Kalamkar; 1st Cl. Sub-Asst. Surg. Mihrab Khan; 2nd Cl. Sub Asst. Surg. Muhammad Kusain; 1st Cl. Sub.-Asst. Surg. Muhammad Riazuddin; 3rd Cl. Sub-Asst. Surg. Mukand Ram; 1st Cl. Sub-Asst. Surg. Nand Lal; 1st Cl. Sub-Asst. Surg. Nur Muhammad; 2nd Cl. Sub-Asst. Surg. Shambhu Nath; 1st Cl. Sub-Asst. Surg. Varyam Singh.

South African Medical Corps.—Capt. A. J. Ballantyne; Temp. Capt. C. P. Bligh Wall; Capt. H. J. Brady; Maj. C. M. Brothers; Capt. R. D. A. Douglas; Maj. (temp. Lt.-Col.) W. V. Field; Maj. (temp. Lt.-Col.) W. Gilbert; Lt.-Col. R. L. Girdwood, D.S.O.; Capt. S. J. O'L. Greensill; Capt. D. Henderson; Capt. J. E. Hurworth; Maj. T. J. W. A. Johnston; Maj. A. Liebaert; Capt. S. MacPherson; Maj. A. D. McKenzie; Capt. H. V. Potter; Capt. W. Shanks; Lt. N. E. Thomson; Capt. G. B. Wilkinson.

East African Medical Service.—Temp. Capt. N. P. Jewell, M.C.; Capt. C. G. Tomblings, African Native Medical Corps; Lt. R. McGeorge; Temp. Capt. J. M. Mackinnon attd. K.A.R.

West African Medical Service.—Temp. Capt. B. J. Courtney; Capt. R. F. Williams, M.C., attd. Gold Coast Regt.

Uganda Medical Service.—Temp. Capt. A. H. Owen; Maj. C. A. Wiggins.

Northern Rhodesia Medical Service.—Capt. J. A. Chisholm; Capt. and Temp. Surg. E. G. Storrs, M.C.

Nyasaland Field Force Medical Service.—Temp. Capt. A. G. Eldred; Temp. Capt. W. C. Wigan.

The names of the following medical officers are mentioned in a despatch dealing with the engagement between the torpedo-boat destroyers *Mary Rose* and *Strongbow* and three German light cruisers which attacked a convoy on Oct. 17th, 1917:—

Surg.-Prob. I. C. C. Barclay, R.N.V.R. (killed in action); Surg. J. P. Shorten, D.S.C., R.N.

OFFICERS OF C.A.M.C. ON H.M.H.S. "LLANDOVERY CASTLE" TORPEDOED.

The following is a brief account of the five officers of the Canadian Army Medical Corps missing, believed drowned, through the torpedoing on June 27th of H.M.H.S. *Llandovery Castle*:—

Lieutenant-Colonel THOMAS HOWARD MACDONALD, who was in command of the ship, was 40 years of age, and a native of Port Mulgrave, Nova Scotia, and graduated at Bellevue Hospital Medical College, N.Y., in 1900. Before the war he was in practice at Port Hawkesbury, N.S., and an active member of the Canadian Militia, in which he held the rank of major. He came overseas in December, 1915, and after various hospital appointments became president of the standing medical board, London area, later serving with a labour battalion in France.

Major GUSTAVUS MITCHELL DAVIS, who was 43 years of age, was a native of York, Ontario, graduating in medicine from the University of Toronto in 1901, and practising at Welland, Ont. He came overseas with the first Canadian contingent in September, 1914. After taking part in the battle of Festubert he was appointed M.O. upon the hospital ship *Letitia*, and when this ship was wrecked on the coast of Nova Scotia in August, 1917, rendered distinguished service in the care and rescue of patients.

Captain ARTHUR VINCENT LEONARD, who was 37 years of age, was a native of Warkmouth, Ontario, graduating in 1911 at the University of Toronto, where he was a prominent athlete. He came overseas in September, 1915, and after serving as M.O. of various units at the front he was appointed to H.M.H.S. *Araguaya*.

Major WILLIAM JAMES ENRIGHT, who was 44 years of age, was a native of Port Daniel, P.Q., graduating in medicine at Laval University in 1897. He served in Canada on the staff of the Quebec Military Hospital before coming overseas. While serving as M.O. in France he was wounded on Sept. 29th, 1915, and evacuated to England, and returned to Canada for three months' sick leave. Later, in charge of embarkation, his name was brought to the notice of the Secretary of State for War. Finally he had been serving as registrar and adjutant of No. 8 Canadian General Hospital.

Captain GEORGE LUTHER SILLS, who was 30 years of age, was a native of Tweed, Ontario. While a student in medicine at Kingston University he joined the C.A.M.C. as a private, serving overseas for a year with the 5th Stationary Hospital. Returning to Canada he graduated in November, 1916.

A QUESTION OF EXEMPTION.

After a conjoined meeting of the Executives of the Edinburgh and Leith Division of the British Medical Association and of the Scottish Medical Guild the following question was sent to Sir Auckland C. Geddes and Sir James Galloway:—

"In the event of a medical man having been granted exemption from ordinary military service on occupational grounds, and called upon to undertake either military or civil work, and he has objections to the work he has been called upon to undertake, will he have the right to appeal to the Medical Tribunal?"

The reply received from Sir James Galloway states that the practitioner has no legal right to a hearing by the Medical Tribunal as such, but that both the Central Professional Committees have arranged that in such circumstances the practitioner would receive a personal hearing, at which his objections to the particular form of service allotted to him would receive every consideration.

THE MEDICAL SERVICE IN THE EAST AFRICAN CAMPAIGN.

—In reply to a question by Commander N. Craig, Mr. Macpherson last week informed the House of Commons that a report by Colonel Pyke and Lieutenant-Colonel Balfour, R.A.M.C., on the Army Medical Service in Africa had been received in May. The report was of a personal and highly confidential nature, and could therefore not be laid upon the table, and effective action had been taken upon it.

OBITUARY OF THE WAR.

FRANCIS METCALFE, M.B., B.S. DURH.,

CAPTAIN, ROYAL ARMY MEDICAL CORPS.

Captain F. Metcalfe, who died on July 10th, was 26 years of age and a medical graduate of the University of Durham. Qualifying in June, 1914, he joined the 1st Northumberland Field Ambulance in the following August. He went to France with his unit in April, 1915, remaining there until he was invalided home in September, 1916, with myelogenous leukæmia. After treatment in the 1st Northern General Hospital he made a seemingly good recovery, and after several months at Catterick returned, at his own request, to France in August, 1917. He went through the March retreat, with all its attendant physical and mental strain, and the leukæmia regained its hold upon him. This time he did not react to treatment, and death occurred after a period of severe dyspnœa in the Framlingham House Hospital for Officers, Newcastle-upon-Tyne. During his college days Captain Metcalfe was good at all field sports and was the heavy-weight champion boxer. As a student he was conscientious in his work, taking good places in class. Retiring in manner and with no outward display, he was felt by his teachers to have qualities which only required time and opportunity to develop. Captain Metcalfe was an only son.

KEYSER ATKIN, B.A. CAMB., M.R.C.S. ENG.,

CAPTAIN, ROYAL ARMY MEDICAL CORPS.

Captain K. Atkin, who died of wounds on June 6th, was only son of Mr. P. W. Atkin, stipendiary magistrate at Salford. He was educated at Mostyn House Preparatory School (where he was head of the school and chief of the cadet corps), at Mill Hill, and Jesus College, Cambridge. On the outbreak of war he applied for a commission, but was told to continue his medical studies and become qualified. This he did in July, 1916, after having acted, when still a student, as house physician at St. Mary's Hospital, and house surgeon at the Royal Infirmary, Manchester. He then at once obtained a commission in the R.A.M.C. attached to the South Staffs Regiment. In September, 1917, when hurrying to the relief of men buried by a shell, he was taken prisoner of war, being released five months later. During his short stay at home he took a special course of dentistry in order to be of greater service to the men on his return to France. While at his dressing-station he received wounds from which he died the same day. Captain Atkin's headmaster writes of him as utterly straight and conscientious, and this reputation remained with him through life. At Mill Hill "he proved himself a boy of great character and stern resolve for right, who made his influence felt for long after he had left." He won distinction as an athlete, rowing in his college boat three times at Henley; rowing was an hereditary accomplishment with him, for his father rowed for Cambridge for three consecutive years. He was also one of the best miniature shots in England, winning the Lord Roberts gold medal; and was second in the Public Schools' boxing competition at Aldershot. At college he was the first man who ever passed the whole of the Second M.B. before taking his B.A. degree, and his teachers predicted for him a splendid future.

JAMES RANDOLPH GYLLENCREUTZ, M.R.C.S. ENG.,

CAPTAIN, ROYAL ARMY MEDICAL CORPS.

Captain J. R. Gyllencreutz, whose death from drowning is now presumed, was in medical command of H.M.H.S. *Salta* mined in the Channel during April of last year. He studied at St. Thomas's Hospital Medical School, taking the conjoint qualification in 1908, and thereafter holding house appointments at the Royal Hospital, Portsmouth, and the Brentford Infirmary. He was then for some years ship surgeon on the Royal Mail line. When war broke out he was senior assistant at the Wandsworth Infirmary. Resigning at once to take a commission in the R.A.M.C., after varied work he went to the Dardanelles in charge of a hospital ship. He went down with his ship, but was picked up by a rescuing vessel which was itself sunk soon after. Captain Gyllencreutz's friends write of his cheerful and breezy disposition. In his own circle he was popular as an amateur actor.

ROBERT NEILSON WALLACE, M.B., CH.B. EDIN.,

CAPTAIN, ROYAL ARMY MEDICAL CORPS.

Captain R. N. Wallace, who died on July 5th in command of a military hospital, was 35 years old and son of the late Dr. R. S. Wallace, of Edinburgh. Graduating in medicine at the University of his native city, he held resident appointments at the Bradford Infirmary and the Manchester Children's Hospital before settling in Edinburgh, where he became clinical assistant to the Ear and Throat Department of the Royal Infirmary. For many years he held a Territorial commission in the R.A.M.C., being attached to the 6th Battalion of the Royal Scots. When called up for permanent duty he qualified as a gas officer and was put in command of the 50th Stationary Hospital at Colchester. While there he took influenza and succumbed to a secondary pneumonia. Captain Wallace was a tall and striking figure. He was widely travelled and possessed of artistic and literary gifts, which accentuate the loss felt by his many personal friends now he is gone.

THE BELL FUND.—Dr. S. A. Kinnier Wilson asks us to acknowledge donations to the Dr. J. H. Bell Fund from Sir Hugh Rigby (£10 10s.), Dr. David Dinwoodie (£1 1s.), Lieut.-Colonel E. Farquhar Buzzard (£5 5s.), and from Captain Ralph R. Watts, R.A.M.C. (£1 1s.). Subscriptions should be sent to Dr. Wilson at 14, Harley-street, London, W. 1.

Medical News.

LONDON (ROYAL FREE HOSPITAL) SCHOOL OF MEDICINE FOR WOMEN.—The following awards of scholarships tenable at the Medical School have been made recently:—St. Dunstan's Medical Exhibition: £60 a year for five years, Miss M. E. Reynolds, North London Collegiate School; £40 a year for five years, Miss M. E. Howard, St. Paul's Girls' School. Isabel Thorne Scholarship, £30, Miss E. A. Perrott, Bromley High School. Sir Owen Roberts Memorial Scholarship, £75 a year for four years, Miss L. G. Iliff, London School of Medicine for Women. Mabel Sharman Crawford Scholarship, £20 a year for four years, Miss D. N. L. Leverkus, London School of Medicine for Women. Agnes Guthrie Dental Bursary, £50, Miss Petronelle Jago, Besheben High School, Brondesbury. Ellen Walker Bursary, £25 a year for two years, Miss G. M. Watkinson, London School of Medicine for Women. Fanny Butler Scholarship, £14 10*s.* a year for four years, Miss A. D. Symons, London School of Medicine for Women.

POST-GRADUATE COLLEGE, WEST LONDON HOSPITAL.—During next week clinics will be held each week-day at 2 P.M., Wednesday, Friday, and Saturday also at 10 A.M.

DEATH OF DR. R. W. SPENCE.—Dr. R. W. Spence, Government medical officer, British East Africa, has died at Nakuru, after an illness extending over ten weeks. Dr. Spence was an active and very capable officer, and was universally trusted in the large district of Nakuru, where he had been stationed since his appointment to the Protectorate Government Medical Service in August, 1913. His death is widely regretted.

LITERARY INTELLIGENCE.—The Oxford University Press announces the forthcoming issue of the first number of "Neurological Studies" from the Seale Hayne Military Hospital, Newton Abbot, edited by Major A. F. Hurst, with the assistance of Captain J. L. M. Symns, Captain W. R. Reynell, and Lieutenant S. H. Wilkinson.

PRESENTATION TO MR. ROBERT BURGESS, L.R.C.P. & S. EDIN., J.P.—Mr. Robert Burgess, who is dispensary medical officer of Coagh Dispensary District, co. Tyrone, and who has been president for a period of 25 years of the Coagh Co-operative Dairy Society, was recently presented with a handsome tea and coffee service by the shareholders of Albany, Coagh, and Curglasson Creameries, and other friends. Mr. Burgess has been largely responsible for the establishment of the creameries in his district of county Tyrone; the Central Coagh Creamery was the first started in the North of Ireland, and did much to re-establish the reputation of Irish butter.

METROPOLITAN HOSPITAL SUNDAY FUND.—At a Council meeting of the Metropolitan Hospital Sunday Fund held at the Mansion House on August 9th, under the presidency of Sir Ernest Tritton, it was announced that the Fund had reached the total of £85,652. The number of participating institutions was 238. The year 1918, said the chairman, was a record one in regard to the amount available for distribution, and this was largely due to a gift of £5000 from the American Red Cross Society. Ministers of religion had carried their appeals into the homes of both rich and poor, either by letters or house-to-house visitation, with very gratifying results, and in one parish £76 had been thus raised in pence alone. Among the largest recipients are the London Hospital £10,217, Guy's Hospital £3467, Middlesex Hospital and Convalescent Home £3730, and St. George's Hospital £3235.

QUEEN'S UNIVERSITY, BELFAST.—At a recent meeting of the Senate it was decided that, for next session, the department of anatomy (vacant through Professor Symington's resignation) should be placed under the direction of Dr. P. T. Crymble, with Dr. R. J. McConnell and Dr. Margaret Purce as demonstrators. It was also arranged to appoint a committee, consisting of the curators and the medical members of the Senate, to confer with the medical faculty in reference to the question of rearranging the work of the Department of Anatomy and to report to the Senate at a later date.—The following have been awarded scholarships in the Faculty of Medicine:—First Medical Examination: W. J. Frackelton, £40; John Beattie, £30; Cecil A. Calvert, £20; and Olive M. Pedlow, £15. Second Medical Examination: S. J. Andrews and J. T. Lewis, £40 each; Eileen M. Hickey, £30; C. H. G. MacAfee, £20; and William S. Gibson, £15. Third Medical Examination: Isaac Eban, Henry Harris, Joseph Meban, and Phillip F. Mooney. Final Medical Examination: William J. Lytle and Henry F. Macauley, medicine, £30 each; Samuel E. Duff and Mary E. Irwin, midwifery, £30 each; Chas. A. Whitfield, surgery, £30; and Charles A. Whitfield, general prize, £20.

Parliamentary Intelligence.

NOTES ON CURRENT TOPICS.

Parliamentary Session.

BOTH Houses of Parliament have adjourned until Tuesday, Oct. 15th.

Royal Assent.

Amongst the measures which received the Royal Assent before the adjournment were the Education Act, the Maternity and Child Welfare Act, and the Asylums and Certified Institutions (Officers' Pensions) Act.

Midwives Bill.

Mr. HAYES FISHER, President of the Local Government Board, has presented in the House of Commons a Bill to amend the Midwives Act, 1902. It was read a first time. The intention is to proceed with it when the House resumes after the autumn adjournment. A Midwives Bill passed through the House of Lords this session, but owing to certain technical obstacles relating to privilege it was not found possible to bring it before the House of Commons. Mr. HAYES FISHER has therefore introduced his measure in substitution for it.

HOUSE OF COMMONS.

WEDNESDAY, AUGUST 7TH.

Indian Medical Service and Children's Allowances.

Answering Colonel YATE, Mr. MONTAGU (Secretary for India) said: Proposals for the grant of children's allowances to officers of the Indian Army, Indian Army Reserve, and Indian Medical Service have been received from the Government of India and are under reference to the War Office. I have proposed that the allowances should be granted with retrospective effect to Oct. 1st, 1917, the date of the commencement of the allowance for British service officers.

Promotion of R.A.M.C. Officers (Territorial).

Sir B. FALLE asked the Under Secretary for War whether he could give any date or information when the promotion of the field officers, Royal Army Medical Corps, Territorial and Volunteer, was likely to take place, or as to the retirement of such officers in default of such promotion.—Mr. MACPHERSON replied: I presume my honourable and gallant friend is referring to the promotions in accordance with the instructions issued recently regarding the introduction of a general seniority list for officers of the Royal Army Medical Corps (Territorial Force). The promotion of the officers of the Territorial Force mentioned will take place as soon as possible after the receipt of the recommendations from the various theatres of war in which these officers are serving. Officers not selected for promotion will not be retired on that account.

Sir B. FALLE: Does that apply to those serving at home?—Mr. MACPHERSON: I presume so.

Asylum Treatment of Lunatic Soldiers.

Replying to Sir WATSON RUTHERFORD, Mr. HODGE (Minister of Pensions) wrote: Men of the Army and Navy whose mental condition has become impaired owing to their service, and who are admitted to asylums, are not separately housed, but special arrangements are made for their maintenance. They are not chargeable to the rates and they do not rank as paupers. Evidence as to mental attack anterior to military service is clearly relevant in determining whether or not the disability is due to service. It is not considered that the provision of separate institutions is practicable, nor is it desirable having regard to the expediency of placing the patients near their friends.

URBAN VITAL STATISTICS.

(Week ended August 10th, 1918.)

English and Welsh Towns.—In the 96 English and Welsh towns, with an aggregate civil population estimated at 16,500,000 persons, the annual rate of mortality was 10·4, against 13·1 and 12·1 per 1000 in the two preceding weeks. In London, with a population slightly exceeding 4,000,000 persons, the death-rate was 10·5, or 0·9 per 1000 below that recorded in the previous week; among the remaining towns the rates ranged from 4·4 in Hornsey, 5·1 in Southend-on-Sea, and 5·3 in Acton, to 17·9 in Gateshead, 18·5 in Rotherham, and 20·3 in Aberdare. The principal epidemic diseases caused 237 deaths, which corresponded to an annual rate of 0·8 per 1000, and included 97 from infantile diarrhœa, 54 from measles, 39 from whooping-cough, 29 from diphtheria, 13 from scarlet fever, and 5 from enteric fever. The deaths from measles and from whooping-cough were the lowest recorded in any week of the current year. The deaths from infantile diarrhœa, which had risen from 34 to 74 in the four preceding weeks, further rose to 97 last week. The 746 cases of scarlet fever and 904 of diphtheria under treatment in the Metropolitan Asylums Hospitals and the London Fever Hospital were 20 and 23 below the respective numbers remaining at the end of the previous week. The 810 deaths from all causes in London included 21 from influenza, against 192, 86, and 38 in the three preceding weeks. The causes of 45 deaths in the 96 towns were uncertified, and included 8 each in Birmingham and in Liverpool, and 6 in Gateshead.

Scotch Towns.—In the 16 largest Scotch towns, with an aggregate population estimated at nearly 2,500,000 persons, the annual rate of mortality was 11·8, against 14·2 and 13·1 per 1000 in the two preceding weeks. The 265 deaths in Glasgow corresponded to an annual rate of 12·4 per 1000, and included 17 from infantile diarrhœa, 6 from whooping-cough, 4 from measles, 2 from diphtheria, and 1 from enteric fever. The 67 deaths in Edinburgh were equal to an annual rate of 10·5 per 1000, and included 1 each from whooping-cough and from infantile diarrhœa.

Irish Towns.—The 117 deaths in Dublin corresponded to an annual rate of 15·3 per 1000, and included 11 from infantile diarrhœa, 2 from measles, and 2 from whooping-cough. The 102 deaths in Belfast were equal to an annual rate of 13·5 per 1000, and included 9 from infantile diarrhœa, 2 from whooping-cough, and 1 from measles.

Appointments.

ASHER, M., L.R.C.S. Irel., has been appointed Assistant County Medical Officer to the Southampton County Council.

AZIZ, A., M.B., Ch.B. Edin., Assistant School and Tuberculosis Medical Officer for Wigan.

BUTTERFIELD, F., M.B. Lond., Temporary Assistant Tuberculosis Officer to the Manchester Corporation.

HOA HING, MARTHA H., L.R.C.P., L.R.C.S. Edin., L.F.P.S. Glasg., House Surgeon at the Alexandra Hospital for Children, Brighton.

PAUL, E. W., M.R.C.S. Eng., Certifying Surgeon under the Factory and Workshop Acts for the East Nottingham District of the county of Nottingham.

TAYLOR, ALICE, L.R.C.P. & S. Irel., L.M., Assistant County Medical Officer to the Southampton County Council.

WILSON, J. M., M.B., Ch.B. Aberd., Certifying Surgeon under the Factory and Workshop Acts for the Aberdare District of the county of Glamorgan.

Vacancies.

For further information refer to the advertisement columns.

Birmingham Education Committee.—Asst. School M.O. £300.
Blackburn County Borough.—Two Asst. M.O.H. £500.
Bramcote Sanatorium, Nuneaton.—Res. M.O. £300.
Bristol Royal Infirmary.—H.P. and H.S. £120.
Bristol University.—Demonstrator in Physiology.
Brook War Hospital, Woolwich, S.E.—Res. M.O. £1 per diem.
Carlisle City, Maternity and Child Welfare.—Asst. M.O. £400.
Cheltenham Eye, Ear, and Throat Free Hospital.—Asst. S. £400.
Derbyshire County Council.—Asst. M.O. £400.
Dorset County Council.—Temp. Female Asst. M.O. £350.
Erdington Infirmary and House.—Asst. M.O. £8 8s. per wk.
Glasgow, Govan District Asylum, Cardonald.—Sen. Asst. M.O. 6 or 7 gs. weekly. Also Locum.
Liverpool, Toxteth Park Guardians.—Asst. Res. M.O. £300.
London County Mental Hospital, Bexley, Kent.—Asst. M.O. 7 gs. wk.
Manchester County Asylum, Prestwich.—L.T. £7 7s. per week.
Manchester Royal Eye Hospital.—Sen. H.S. £120.
Middlesex Education Committee.—Asst. School M.O. £400.
Newcastle-upon-Tyne Education Committee.—Asst. School M.O. £350.
Norwich, Norfolk and Norwich Hospital.—H.S. £400.
Nottingham General Hospital.—Two Female H.S. £250.
Notts Education Committee.—Asst. Sch. M.O. £400.
Queen Mary's Hospital, Stratford.—H.S.
Rochdale Infirmary and Dispensary.—Jun. H.S. £150.
Sheffield Royal Infirmary.—Res. S.O. £200. Also H.S. £150.
Stoke-on-Trent County Borough.—Asst. Tuberc. O. £400.
Tottenham Urban District Council.—M.O. for Maternity and Child Welfare Centre.
Walsall General Hospital.—Female H.S. and Anæs. £175.
Westmorland Consumption Sanatorium.—Asst. M.O. £200 to £250.
Whitehaven and West Cumberland Infirmary.—Res. H.S. £150 to £180.

Births, Marriages, and Deaths.

BIRTHS.

BREWERTON.—On August 6th, at Harley-street, the wife of Elmore Brewerton, F.R.C.S., of a son.

HUNT.—On August 10th, at Cahermoyle, South Farnborough, Hants, the wife of Ernest Rivaz Hunt, M.D., M.R.C.P., Temp. Capt., R.A.M.C., of a daughter.

MCKEE.—On July 25th, the wife of Surgeon A. G. McKee, R.N., of a son.

PATTISON.—On August 10th, at Hollow Meadows, Sheffield, Pamela, the wife of C. Lee Pattison, M.B., B.S. Lond., of a son.

RICHARDS.—On August 9th, the wife of Lieutenant J. E. Richards, R.A.M.C., of a daughter.

MARRIAGES.

ADDINSELL—KERRISON.—On August 8th, at St. George's, Hanover-square, Augustus W. Addinsell, M.B., M.R.C.P., to Mrs. Diana Kerrison, of Tattingstone.

DEATHS.

DERRY.—On August 8th, at St. Leonard's House, Bodmin, Bartholomew Gidley Derry, M.R.C.S. Eng., L.R.C.P. Lond., aged 71 years.

LLOYD.—On August 4th, killed in action, Captain W. H. Lloyd, M.B., B.S. Lond., M.R.C.S., L.R.C.P., aged 27.

NIALL.—On August 7th, at Dulwich-road, S.E., Eugene Hayes Niall, L.R.C.P. & S. Edin., aged 76 years.

WALTERS.—While on a visit to Ashbrook Hall, Hollington, Sussex, John Basil Walters, M.R.C.S., L.R.C.P.

N.B.—A fee of 5s. is charged for the insertion of Notices of Births, Marriages, and Deaths.

BOOKS, ETC., RECEIVED.

BAILLIÈRE, TINDALL, AND COX, London.
Aids to Medical Diagnosis. By A. Whiting. 2nd ed. 3s.
Errors of Accommodation and Refraction. By E. Clarke, M.D. 6s.

CHURCHILL, J. AND A., London.
Treatise on Applied Analytical Chemistry. By Prof. V. Villavecchia and Others. Translated by T. H. Pope, B.Sc. Vol. II. 25s.
Short Practice of Midwifery for Nurses. By H. Jellett, M.D. 5th ed. 8s. 6d.

LEWIS, H. K., London.
Statics of the Female Pelvic Viscera. Vol. I. By R. H. Paramore. 18s.
The Twin Ideals: An Educated Commonwealth. By J. W. Barrett, K.B.E., Temp. Lieut.-Col., R.A.M.C. 2 vols. 25s.

LONGMANS, GREEN, & Co., London.
Colour in Relation to Chemical Constitution. By E. R. Watson, D.Sc. 12s. 6d.
Commonwealth Book of Cookery. By Muriel V. Palmer. 2s. 6d.
Modern Treatment of Nervous Disorders. By Bernard Hart, M.D. 1s.
Coal and Its Scientific Uses. By W. A. Bone, D.Sc., F.R.S. 21s.
Elements of the Electromagnetic Theory of Light. By Ludwick Silberstein, Ph.D. 3s. 6d.

MASSON ET CIE., Paris.
Précis de Dermatologie. Par le Dr. Darier. 18 fr.
Études sur le fonctionnement rénal dans les néphrites chroniques. Par le Dr. Pasteur Vallery-Radot. 10 fr.
Commotions et Emotions de Guerre. Par Professeur André Léri, Préface du Professeur Pierre Marie. 4 fr.
La suspension dans le traitement des fractures. Par M. P. Desfosses et le Dr. Charles-Robert. Préface de M. Pierre Duval. 4 fr.

MURRAY, JOHN, London.
Sketches of the East Africa Campaign. By Capt. R. V. Dolbey, R.A.M.C. 6s.

OXFORD PRESS WAREHOUSE, London.
The Hearts of Man. By R. M. Wilson, M.B. 6s.
Gymnastic Treatment for Joint and Muscle Disabilities. By Brevet Col. H. E. Deane, R.A.M.C. With preface by Col. A. Carless, A.M.S., and by Brevet Lieut.-Col. F. W. Mott, F.R.S., R.A.M.C. (T.F.).

SAUNDERS (W. B.) COMPANY, Philadelphia.
Treatise on Clinical Medicine. By W. H. Thomson, M.D. 2nd ed. 24s.
Local and Regional Anæsthesia. By C. W. Allen, M.D. 28s.
Chemical Pathology. By H. G. Wells, M.D. 19s.
Treatment of War Wounds. By W. W. Keen, M.D. 2nd ed. 8s. 6d.

WRIGHT, JOHN, AND SONS, Bristol.
War Wounds of the Lung. By P. Duval. 8s. 6d.

The following journals, magazines, &c., have been received:—Indian Journal of Medical Research, Veterinary Review, Canadian Medical Association Journal, Journal of Hygiene, Journal of State Medicine, National Food Journal, Journal of Comparative Pathology and Therapeutics.

EDITORIAL NOTICE.

In consequence of the pressure on our space and the shortage of paper, the Editor may be compelled to abbreviate communications, as an alternative to their rejection, when time does not allow them to be reconsidered by their authors.

Communications, Letters, &c., to the Editor have been received from—

A.—Major A. Abrahams, R.A.M.C.; *Answers*, Lond., Editor of; Mr. R. J. Albery, Lond.; Miss L. G. Ackroyd, Bramhall.
B.—Miss C. S. Bremner, Crosthwaite; Board of Education, Lond., Sec. of; Capt. W. Langdon Brown, R.A.M.C.(T.); British Science Guild, Lond.; Messrs. Butterworth and Co., Calcutta; Pte. A. G. Bedane; Mr. J. Brindley-James, Lond.; Mr. S. Bhatta; Dr. H. W. Bywaters, Bristol; Major W. Brown, R.A.M.C.
C.—Capt. J. Campbell, R.A.M.C.; Mrs. E. S. Chesser, M.B., Lond.; Mr. J. Cabburn, Lond.; Dr. E. L. Collis, Lond.; Dr. W. M. Crofton, Dublin.
D.—Dr. W. R. Dunstan, Lewes; Capt. A. D. Dunne, R.A.M.C.(T.); Capt. S. C. Dyke, R.A.M.C.; Lieut.-Col. A. M. Davies, R.A.M.C.
E.—Major R. Eager, R.A.M.C.(T.); Ex-Officers Employment Bureau, Lond.
F.—Mrs. S. E. Foott, Cork; Capt. C. D. Farquharson, C.A.M.C.
G.—Mr. M. Gault, Limavady; Dr. H. L. Gordon, Lond.; Mr. F. C. Goodall, Lond.
H.—Mr. A. Harden, Lond.; Dr. J. H. Hart, East Molesey; Capt. E. Hindle, R.E.
J.—Dr. G. Jessel, Wigan.
K.—Major M. F. Kelly, R.A.M.C.; Mr. W. H. Kesteven, Kingston Hill; Dr. D. Keilin, Cambridge.
L.—London (Royal Free Hospital) School of Medicine for Women, Warden of; Dr. R. B. Low, Lond.; Dr. T. Lumsden, Lond.; Mr. E. P. Leavy, Innisfail; Messrs. Lea and Febiger, Philadelphia.
M.—Dr. D. L. Mackinnon, Aberdeen; Malay States Information Agency, Lond.; Mr. W. B. McCall, Birmingham; Mr. J. E. R. McDonagh, Lond.; Metropolitan Asylums Board, Lond.; Ministry of Munitions, Director of Special Intelligence, Lond.; Capt. J. C. McWalter, R.A.M.C.
N.—National Physical Laboratory, Teddington.
O.—Dr. H. C. Orrin, Lond.
P.—Dr. H. W. Pooler, Alfreton; Dr. E. E. Prest, New Cumnock.
R.—Dr. H. S. Reynolds, Wisbech; Mr. P. B. Roth, Lond.; Dr. H. Rahmet Bey, Cairo.
S.—Capt. R. Steele, R.A.M.C.; Summer School of Civics and Eugenics, Lond., Sec. of; Dr. H. Sutherland; Dr. G. E. Shuttleworth, Bournemouth; Mr. W. Scott, Bridgwater.
T.—Dr. J. Tatham, Oxted; Capt. W. J. Tulloch, R.A.M.C.; Mr. H. B. Tawse, Nottingham; Capt. A. G. Tressider, I.M.S.
W.—Mrs. M. B. Wilson, Petworth; West Sussex County Asylum, Chichester; Dr. P. L. Watkin, Williams.

Offices: 423, STRAND, LONDON, W.C. 2.

Notes, Short Comments, and Answers to Correspondents.

COLONIAL HEALTH REPORTS.

Nyasaland.—A report prepared by Mr. G. B. Anderson, acting Assistant Chief Secretary, dealing with the year ending March 31st, 1917, states that during that period the public health was not as satisfactory as heretofore owing to military requirements for labour having necessitated an abnormally increased movement of natives in all parts of the territory.

"Small-pox," Mr. Anderson records, "was still confined to the Lilongue and Dowa districts, and although labour was recruited from both these areas the precautionary measures which were instituted proved successful in preventing any spread of the disease. Two localised outbreaks which occurred in the Zamba and Upper Shire districts respectively among labourers employed on the military roads were promptly and effectually dealt with. As in the previous year, sleeping sickness investigations were confined to the proclaimed area in the Dowa district, the number of cases reported amounting to 31. In 1915 the figures were 29 from this area, plus 4 from other parts of the Protectorate, making a total of 29. It would be misleading to assume that these figures represent the actual incidence of sleeping sickness in the Protectorate as a whole, for the results of investigations in the past have shown that the portion of the Yarimba district bordering on the lake is as heavily infected as the proclaimed area in the Dowa district. It is much to be regretted that bubonic plague has made its appearance in the Protectorate for the first time. Every effort was made to ascertain the source whence this disease gained entry into the North Nyasa District, and inquiry has shown that there can be little doubt but that it was introduced from German East Africa during the latter part of the year, when there was considerable movement of the native carriers between Karonga and Yuraya. Three cases of bubonic plague were reported in the neighbourhood of Karonga in the month of November, 1916, and ten in December. Further cases occurred during the following months, but at the moment of writing it is satisfactory to record that following the campaign of rat extermination, inoculation, and the adoption of other preventive measures, the outbreak is now well in hand and the disease has not shown any indication of spreading to other parts of the Protectorate. The number of Europeans resident in the Protectorate, including officials and non-officials, was 474 males and 257 females, or a total of 731, as compared with 523 males and 262 females, or a total of 785, in the previous year. The Asiatic population amounted to 391, as against 379 in the preceding year. The native population is estimated at 1,137,266, being approximately 506,785 males and 630 481 females."

British Honduras.—A report furnished to the Colonial Office by Mr. W. L. McKinstry, Acting Colonial Secretary of British Honduras, states that the estimated mean population for the year 1916 was 42,323, consisting of 21,101 males and 21,222 females. The following table shows the number of deaths for every 100 births registered during the years 1911–15 inclusive, according to their race classification, and as compared with 1916:—

Race.	Deaths for every 100 births, 1911–15.	Deaths for every 100 births, 1916.
Indian	80·76	100·5
Carib	52·05	43·79
Negro	53·89	59·51
Other races	63·51	57·99

The birth-rate was 39·056 and the mortality 26·321 per 1000 of the estimated mean population. The number of illegitimate births was 687 out of a total of 1653, or 41·56 per cent. 413 marriages took place during the year, being at the rate of 9·75 per 1000 of the mean population. During 1916 the excess of female over male births was 85; and of male over female deaths 150. The colony suffered sudden and severe losses in the untimely deaths of Lieutenant-Colonel Slack and P. C. Burgess, at the hands of a murderer, and Dr. J. H. H. Harrison, the principal medical officer, at the hands of a lunatic. The deepest sympathy of all classes was extended to the bereaved families. The health of the colony had been on the whole good. Slight epidemics of dengue, measles, influenza, and chicken-pox occurred, but were all of a mild type. Infantile mortality was unfortunately very high in most districts. During the first six months of the year 1916 Dr. Hackett, of the Rockefeller Institute, conducted an inquiry into the prevalence of ankylostomiasis throughout the colony. He found an infection varying from practically nil on the Cayes to nearly 100 per cent. in some of the Indian villages. Determined steps were taken to combat the disease.

Northern Territories of the Gold Coast.—In his latest report on this Protectorate Mr. C. H. Armitage, the Chief Commissioner, states that two deaths occurred among the European officials—one from heart failure in February at Tamale and one from yellow fever at Gambaga in October. The names of 12 officials appeared on the sick list. Among the native officials only 16 entries occurred on the sick report, with one death. The Census, completed in 1911, gave an estimated native population of 361,806 to the Northern Territories, with a computed area of 31,100 square miles, an average of 11·6 persons to the square mile. The Census returns for the Southern and North-Western Provinces were considered to be fairly accurate, but those of the North-Eastern Province fell far short of being correct, and it is estimated that not more than two-thirds of the inhabitants were enumerated. In spite of the heavy infant mortality it is considered that the population at the present day is greatly in excess of that shown in the last Census returns. Except for an outbreak of small-pox at Wa at the end of the year, no epidemic disease was recorded. It is reported that the Walas inoculated their children from the virus obtained from those who contracted small-pox. Two deaths from anthrax occurred at Salaga. Leprosy is not on the increase, and, while uncommon in the Southern and North-Western Provinces, is of more frequent occurrence in the North-Eastern Province. The natives look upon it as "a visitation of Allah," but say that it shows no signs of spreading, and are generally averse to compulsory isolation. In concluding his report, Mr. Armitage remarks that:—

"The climate of the Northern Territories, while similar to that of the Gold Coast and Ashanti, may be said in comparison with them to be more trying in its extremes. It possesses an insidious quality that lowers the vitality of Europeans and renders unduly prolonged residence on their part inadvisable, and it is generally recognised that a period of recuperation in a more bracing and salubrious climate is necessary after a year's stay in the country. Innumerable insects—creeping and flying—are a source of irritation, especially at evening time and after dark."

BORO-CHLORETONE IN THE TREATMENT OF BURNS.

To the Editor of THE LANCET.

SIR,—I should like to draw the attention of those of your readers who have not yet tried it to the value of this powder in the treatment of burns and scalds. I have used it for several years and have found it most efficacious, and its marked success in a recent case of a young girl, whose leg and foot were badly scalded, was most noticeable. I found her screaming with pain. Within five minutes of a copious dusting of the damaged skin with boro-chloretone all pain had disappeared and she was able to sleep the whole night. The next day the circumvesicular erythema had distinctly faded, and healing rapidly ensued. Boro-chloretone not only anæsthetises the skin but diminishes the inflammation, and being in addition an antiseptic fulfils the qualifications of an ideal dressing for burns and scalds.

I am, Sir, yours faithfully,

Watton, Norfolk, August 4th, 1918. H. MALLINS.

MEMORIAL TO THE LATE MR. R. H. GRIMBLY, O.B.E., M.R.C.S., L.S.A.

ON July 11th, at Newton Abbot Ambulance Hall, a memorial tablet was unveiled to the memory of the late Mr. Richard Henry Grimbly, who was the founder of the local corps of the St. John Ambulance Brigade.

EGG "SUBSTITUTES."

AN editorial article in the *Journal of the A.M.A.* for July 13th has some hard things to say about the numerous products at present vaunted in America as efficient substitutes for the hen's egg. At the request of the Pennsylvania State Department of Agriculture, Professor C. H. Lawall has investigated more than 40 of these substitutes, and in the introduction to his report he says:—

One of the most reprehensible ways of making money is to take some common, everyday substance, disguise or alter its appearance in some way, make a lot of exaggerated statements regarding it, and then sell it for about 10 or 15 times its market value, extolling it as an economical substitute for some expensive article. There has been no preparation of this class within recent years that has sprung into prominence with such rapidity as the so-called egg substitutes and with so little merit or legitimate warrant for their manufacture and sale.

Practically all the substitutes have been found to consist chiefly of cornstarch coloured with coal-tar dye. The protein content varied from 0·7 to 35 per cent. Commercially dried egg, which is a legitimate market product, contains about 45 per cent. of protein. Professor Lawall concludes that all so-called egg substitutes afford a ready opportunity for profiteering, combined with the development of the art of camouflage to the point of perfection.

"Their names are deceptive; their composition in no wise resembles that of egg; the presence of colour, in those where it is used, is a fraud, and the claims as to replacing value are either deliberate misstatements or ambiguous phrases. Egg substitutes serve no purpose that cannot be served just as satisfactorily and much more cheaply by articles in daily use in every household."

The egg "substitutes" are probably similar to what are known in Great Britain as custard powders, which are presumed to make "custard without eggs." In this country there is no legal definition laying down a standard, so far as we know, for such powders, although in Pennsylvania such substitutes contravene the State food regulations, and if found for sale are dealt with accordingly. As was shown in a report from THE LANCET Laboratory in 1900 (THE LANCET, May 5th, 1900, p. 1298) the powders consist practically entirely of cornstarch coloured with turmeric or coal-tar dye. They have therefore nothing in common with eggs, and the "custard" made from them is merely a starch emulsion with very small real nutrient value.

SOME MEDICAL NOTES FROM ANCIENT MESOPOTAMIA.

Dr. Paul Haupt, of Johns Hopkins University, has, in the *United States Oriental Research Journal*, been giving a revised translation of the difficult cuneiform text in the Annals of Assurbannipal, king of Assyria, describing the illness of Teumman, king of Elam, with whom the Mesopotamians were at war. This record has been for the last 40 years differently rendered, and was thought to indicate that the disease was rabies, because one sentence is capable of being read as "he behaved like a mad dog"; but Professor Haupt, after an elaborate discussion of the text from our knowledge, now so advanced, of the cuneiform vocabulary, and a comparison of the words with their congeners in other Semitic dialects, shows that the following is the correct translation. "At that time he (Teumman) had an attack, his lip slavered, his eye rolled; wildness was imported to it." This version agrees quite closely with the malady being an epileptic fit, for during an attack of morbus sacer the eyes roll wildly and the sufferer foams at the mouth. The ancients were agreed that such disease was specially inflicted by the gods, and in a further part of the inscription the origin of it is attributed to Assur and Ishtar.

The existence of veterinary surgeons in very early times in Babylonia is disclosed by the ancient law code of King Hammurabi. This is confirmed by a cuneiform tablet, Rm. 362 in the British Museum, which, though much defaced, scholars can detect gave a list of plants useful for treating colic in the horse. One line reads, "Plant for abdominal cutting pain in the horse." Other lines read in Babylonian what is most nearly translated by "Contunde in vino, ungue ad abdomen." This tablet probably presents the earliest known instance of cataplasmata being utilised for treatment of colic of the horse.

Dr. Felix von Oefele, whose residence appears now to be in New York, has been writing in the *Journal of the American Oriental Society* upon a Babylonian statuette of the jerboa, or Egyptian jumping mouse, especially with regard to the accurate representation of zoological details showing careful morphological observation. The double length of the tibia in comparison with the femur is carefully copied. The animal is still to be found in the Western Babylon desert, and doubtless it was there, and not in the Sinai peninsula, that the artist obtained the specimen he copied.

"VITAMOGEN."

To the Editor of THE LANCET.

SIR,—My attention has been called to a leaflet composed of extracts from an article of mine on Vitamines published in *Science Progress*, 1914, followed by references to a food preparation called "Vitamogen" purporting to contain these accessory growth substances.

The publication of my name in connexion with this matter is entirely without my sanction, and should not be taken to imply that I consider that there are any vitamines in the above-mentioned preparation.

I am, Sir, yours faithfully,

Bristol, August 12th, 1918. H. W. BYWATERS, D.Sc.

*** We have also received a communication from Vitamogen, Ltd., in which they enclose a copy of a letter which this company wrote to Dr. Bywaters on his drawing their attention to the matter. The letter is as follows:—

[COPY.]

Professor H. W. Bywaters, July 31st, 1918.
c/o Messrs. Fry and Sons, Ltd., Bristol.

DEAR SIR,—We have yours of the 24th inst. addressed to Mr. Shepperson, and we are very sorry you feel you have cause for grievance against us.

We assure you that we had no intention of doing anything to cause you to feel any disturbance of mind, as we have acted quite innocently in the matter, inasmuch as, after Mr. Shepperson's interview with you at Bristol, on your suggestion he communicated with Mr. Murray regarding the loan of the blocks, and at the same time mentioned that we should like to publish extracts from your paper and his reply was, that "provided the usual acknowledgment is made of the source from whence it is taken." This is from his letter which we have before us at the moment, dated Oct. 29th, 1917, and you will observe we have done this, and a proof of our leaflet was sent to the publishers of *Science Progress* for perusal before we issued it.

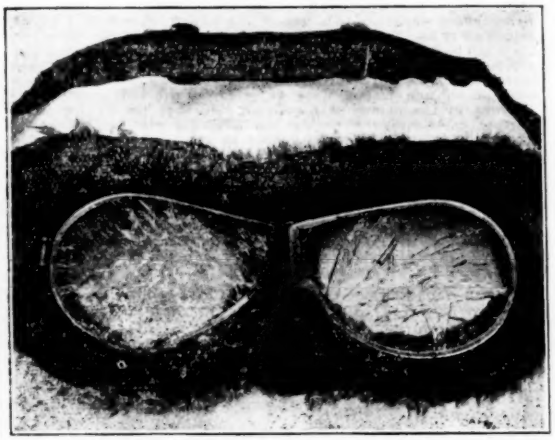
We remain, yours faithfully,
VITAMOGEN, LTD.

HOUSEHOLD ECONOMY.

THREE recently published little books will greatly help towards household economy in regard to the preparation and preservation of food. The first, *The Commonwealth Cookery Book*, by Muriel V. Palmer (London: Longmans, Green, and Co. Price 2*s.* 6*d.*), contains within the compass of 124 pages a large number of recipes, the result of many years of experience, the author being organiser of domestic subjects to the Birmingham Education Committee. This thoroughly practical little book gives instructions for the preparation of every-day dishes, including the requirements of invalids, and for the simple preservation of fruits and vegetables. A recipe supplied by Messrs. Cadbury Bros. for a cocoa-butter emulsion makes for economy in cake and biscuit making.—*Canning and Bottling*, with notes on other simple methods of preserving fruits and vegetables, by Helen P. Goodrich, D.Sc., late technical adviser on canning in the Food Production Department (Same publishers. Pp. 70. Price 2*s.*), deals with the subject in a very simple and thorough manner, the first part presenting all the information necessary for the successful preservation of fruits and vegetables, the last part being devoted to more technical and theoretical matters interesting to those proposing to can on a large scale. The author believes "that the saving of fruit and vegetables may not only be effected during the war, but, through the lessons in economy that we are learning now, a pleasant and financially sound occupation may be fostered as a permanent national village industry, which cannot but make agricultural life more attractive."—An enlarged edition of Miss Helen Wyldon's *Sugarless Jam Making*, published at 10, Glendale-terrace, Bideford, Devon, price 1*s.* 2*d.*, post free, also contains instructions on preserving fruit and vegetables as well as bottling and drying, corn syrup preserving, together with hints for canners. At a time when there is a shortage of sugar this little book, with a score of recipes, should prove useful.

TRIPLEX GLASS.

Mr. Charles Higgens recently called attention in this column to the properties of this glass and the protection which it offers to the eyes of our fighting men. We have received from the Triplex Safety Glass Co., Ltd.



(1, Albemarle-street, W. 1), a photograph, which we reproduce here, of a pair of safety-glass goggles which stood a severe test. A lieutenant in the R.A.F. writes that he was wearing the goggles when he crashed, and that they were the means of saving his eyesight.

MATERNITY FEES IN EAST RIDING.

THE county medical officer has reported to the Health Committee of the East Riding county council that he has been in communication with the acting secretary of the East Riding local medical committee with regard to the fees to be paid by the county council under the maternity and child welfare scheme to practitioners who were summoned in necessitous cases by a midwife employed by the council, and also to practitioners who attended, under arrangements made by the council, necessitous women at confinement. The fees suggested by the local medical committee were as follows:—

1. Where the practitioner is called in by the midwife in a necessitous case, and the distance is within three miles, not exceeding £2 2*s.*; where the distance is more than three miles, not exceeding £3 3*s.* The fee for subsequent visits to be 3*s.* 6*d.*, plus mileage of 1*s.* a mile or part of a mile one way.

2. Where the practitioner undertakes the case by arrangement with the council:—

	£	s.	d.
Where the distance is a mile or under	1	11	6
" " over one mile and under three miles	2	2	0
" " three miles and over	2	12	6

The committee approved of this scale.

SOMERSET CENTENARIANS.

Mrs. Russell, of Weston-super-Mare, celebrated the 100th anniversary of her birthday on July 19th. Weston-super-Mare has had four centenarians within the past four years.—Mr. F. W. Stabbins, of Worle, was 103 years of age on July 18th.

J. S. McW. has omitted to give the reference for the statement which he quotes.